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CONTENTS OF VOLUME VIII

JULY, 1914. NUMBER 1

PAGE

STUDIES IN THE RESPIRATORY EXCHANGE OF INFANTS. FRANCIS G. BENEDICT AND FRITZ B. TALBOT, BOSTON 1

A CASE OF ACUTE CHYLOUS ASCITES (NON-FATTY, PSEUDOCHYLOUS, LACTESCENT OR MILKY TYPE) IN A BOY EIGHT YEARS OLD. FRANCIS HUBER, M.D., NEW YORK. OPERATIONS AND SURGICAL COMMENTS BY HENRY M. SILVER, M.D., NEW YORK 50

PROGRESS IN PEDIATRICS: REVIEW OF THE LITERATURE ON THE FEEDING AND GASTRO-INTESTINAL DISEASES OF INFANTS, FOR THE YEAR 1913-1914, GRACE MEIGS, M.D., CHICAGO 58

REVIEW OF SYPHILIS WITH ESPECIAL RELATION TO HEREDITARY SYPHILIS. HARVEY PARKER TOWLE, M.D., BOSTON 95

INDEX TO CURRENT PEDIATRIC LITERATURE 106

AUGUST, 1914. NUMBER 2

PAGE

PRIMARY PULMONARY ACTINOMYCOSIS IN A CHILD AGED TEN, WITH POST-MORTEM EXAMINATION. FRANCIS HUBER, M.D., AND SAMUEL BERKOWITZ, M.D., NEW YORK 113

CASEIN IN INFANT-FEEDING: THE PREPARATION OF DRY POWDERED PARACASEIN AND PRELIMINARY EXPERIMENTS CONCERNING ITS USE AS THE PRINCIPAL PROTEIN CONSTITUENT IN INFANT FOOD. A. W. BOSWORTH, A.M., GENEVA, N. Y., AND HENRY I. BOWDITCH, M.D., BOSTON, WITH THE ASSISTANCE OF B. H. RAGLE, A.B. 120

THE PRESENCE OF LACTIC ACID IN THE URINE IN CYCLIC VOMITING OF CHILDHOOD. FRANK P. UNDERHILL AND H. MERRIAM STEELE, M.D., NEW HAVEN, CONN. 127

A CLINICAL STUDY OF TYPHOID FEVER IN CHILDREN. HENRY DWIGHT CHAPIN, M.D., NEW YORK 130

DIAGNOSIS OF WHOOPING-COUGH BY THE COMPLEMENT-DEVIATION TEST. ALFRED FRIEDLANDER, M.D., AND E. A. WAGNER, M.D., CINCINNATI 134

THE ARTIFICIAL COOLING OF SICK-ROOMS IN SUMMER. A PRELIMINARY REPORT. HENRY HEIMAN, M.D., NEW YORK 138

CIRCUMCISION IN THE MASTURBATION OF FEMALE INFANTS. ROWLAND G. FREEMAN, M.D., NEW YORK 144

MIDDLE EAR COMPLICATIONS OF MEASLES IN IMMIGRANT CHILDREN. H. C. CODY, M.D., U. S. PUBLIC HEALTH SERVICE, ELLIS ISLAND 147

A CASE OF TUBERCULOUS MENINGITIS COMPLICATED BY INFLUENZAL MENINGITIS. H. K. FABER, M.D., NEW YORK 150

PROGRESS IN PEDIATRICS: ACTIVE IMMUNIZATION AGAINST DIPHTHERIA BY MEANS OF VON BEHRING'S VACCINE, AND THE DIPHTHERIA TOXIN SKIN REACTION. BORDEN S. VEEDER, M.D., ST. LOUIS, MO. 154

DIGESTIVE DISTURBANCES OF ARTIFICIALLY FED BABIES. IRVING M. SNOW, M.D., BUFFALO 163

INDEX TO CURRENT PEDIATRIC LITERATURE 178

American Journal of Diseases of Children

Vol. 8 OCTOBER, 1914 No. 4

BLOOD-PRESSURE IN NORMAL CHILDREN*

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In pediatrics the amount of research in blood-pressure has been limited, and so far, but little of real value has been developed. While blood-pressure determination in adults is conceded to be of inestimable value, many practitioners consider that it has little or no significance in children. This statement is not reasonable unless we assume that there are many additional factors, especially in technic, interfering with accurate readings, and that such wide variations are encountered as to vitiate the results.

We have undertaken a series of studies in normal children, in order to determine whether a standard of blood-pressure could be established. Could such a standard be made, it would open to the pediatrician a new aid in diagnosis and prognosis and help in the treatment of a large number of diseases. Blood-pressure readings will also furnish a means of determining the physical tone of a large number of children who are below par but have no demonstrable pathologic lesion. The whole problem depends on our having an accurate and reliable means of obtaining the readings. After the attainment of a correct technic a sufficient number of observations should be taken in order to establish a standard.

The blood-pressure of children, as of adults, depends on four main factors: the contracting force and rate of the heart; the peripheral resistance of the arterioles and capillaries; the elasticity of the vessel walls; and the character of the blood as to volume, viscosity, etc.

The contracting force and rate of the heart is the most important factor. Owing to the relatively larger, more elastic, and distensible arterioles and capillaries found in children, the peripheral resistance is less marked than in adults, and the vessel-walls are in a less stable state of equilibrium. This simply means that the systolic blood-pressure is a more direct representation of the work of the heart than in the adult. The importance of the vasomotor system must not be overlooked as one of the main factors causing variations in blood-

*Read before the Section on Diseases of Children at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

pressure in childhood. The volume and viscosity of the blood are as a rule of slight importance.

All blood-pressure determinations are influenced by certain physiologic factors. The systolic blood-pressure varies with the increasing age of the child. Blood-pressure changes with alterations in weight and, in children of the same age, varies according to height. The influence of sex is less than in the adult, with slightly higher readings in the males. Emotions cause an increase in pulse-rate and a consequent elevation of blood-pressure. Within fifteen or twenty minutes after meals there is a rise, and a fall again in three-quarters of an hour to an hour and a half.[1] There is a gradual fall in blood-pressure during sleep.[2] At the end of the day blood-pressure is higher than in the morning. There is a rise in blood-pressure following exercise, directly in proportion to the amount of muscular effort. During inspiration blood-pressure falls, to rise again during expiration. In healthy children breathing does not exert much influence except during prolonged forced and deep respiration.

Since there are more or less constant changes in blood-pressure from day to day, it is important in children, as in adults, to secure a series of observations on each individual.

Among the earlier American workers in this field was Cook,[3] who used a modified Riva-Rocci instrument with a narrow 5-cm. cuff, and took observations of the systolic pressure only, using the palpation method. He did not report any diastolic readings. Stowell and Lennox Gordon[4] have also reported systolic blood-pressure readings from a number of cases, using a 12-cm. cuff, and a mercurial instrument. Investigations on fifty-eight children were carried out by N. Oppenheim and S. Bauchwitz,[5] using a modified Riva-Rocci apparatus with a 5-cm. cuff. They made no determinations of the diastolic pressure. A large series of observations on children by the palpation method were made by W. Kaupe,[6] using a Riva-Rocci instrument with a 12-cm. cuff. He took as the systolic pressure the average reading at the time of disappearance and reappearance of the radial pulse to the palpating finger. The method of obtaining diastolic readings is not stated.

In Sahli's clinic an extended series of blood-pressure observations was made by P. Wolfensohn-Kriss,[7] using a Riva-Rocci instrument

1. Loeper: Arch. d. mal. du coeur, 1912, p. 225.
2. Brush and Fairweather: Am. Jour. Physiol., 1901, p. 199; Brooks, Harlow, and Carroll, John H.: A Clinical Study of the Effects of Sleep and Rest on Blood-Pressure, Arch. Int. Med., August, 1912, p. 97.
3. Cook: Am. Jour. Med. Sc., 1903, cxxv, 433.
4. Stowell and Gordon, Lennox: Arch. Pediat., May, 1911.
5. Oppenheim, N., and Bauchwitz, S.: Arch. f. Kinderh., 1905, xlii, 415.
6. Kaupe, W.: Montschr. f. Kinderh., 1910-11, ix, 257.
7. Wolfensohn-Kriss, P.: Arch. f. Kinderh., 1910, liii, 332.

TABLE 1.—Systolic, Diastolic and Pulse-Pressure in Normal Children by Three Methods—Authors' Observations *

Years	Width Cuff cm.	No. of Observation	Circ. of Arm cm.	Pith-Ball			Auscultation			Erlanger Mod.			Pulse
				S. P.	D. P.	P. P.	S. P.	D. P.	P. P.	S. P.	D. P.	P. P.	
3	9	24	13.5-14.0	88.8	54.1	34.7	92.0	D 58.4 G 41.6	33.6	91.8	65.6	26.2	92
4	9	95	13.0-16.5	93.7	59.8	33.9	92.6	D 61.7 G 49.1	30.9	91.6	64.9	26.7	99
5	9	69	13.0-18.0	97.4	61.1	36.3	91.6	D 60.0 G 46.0	31.6	91.3	64.4	26.9	93
6	9	110	13.0-16.0	96.1	62.7	23.4	93.8	D 63.5 G 51.9	30.3	92.6	67.3	25.3	95
7	9	145	14.5-17.5	99.3	64.4	34.9	87.9	D 64.2 G 49.2	22.7	94.4	66.4	28.0	87
8	13	128	15.0-20.0	93.7	64.6	29.1	93.0	D 59.6 G 41.0	33.4	93.6	64.7	28.9	88
9	13	149	16.0-19.0	103.3	70.6	32.7	91.7	D 62.2 G 50.0	29.5	94.3	71.0	23.3	84
10	13	203	15.0-19.5	100.2	65.7	34.5	99.0	D 64.6 G 47.3	34.4	99.2	67.1	32.1	87
11	13	169	15.0-20.5	98.5	74.4	24.1	95.8	D 62.3 G 49.1	33.5	97.1	65.6	31.5	87
12	13	94	17.0-24.0	100.9	64.0	36.9	99.9	D 59.6 G 37.6	40.3	102.3	65.2	37.1	89
13	13	80	14.0-22.5	103.0	66.0	37.0	104.0	D 63.2 G 47.0	30.8	103.6	70.5	33.1	96
14	13	43	18.0-25.0	109.7	69.4	40.3	105.8	D 63.7 G 48.2	42.1	106.1	67.4	38.7	84
15	13	35	17.5-24.0	103.2	68.5	34.7	99.6	D 61.8 G 43.6	37.7	105.6	67.5	38.1	84

* S. P. = systolic pressure; D. P. = diastolic pressure; P. P. = pulse pressure; D. = beginning dull or fourth phase; G. = all sound gone, fifth phase.

with a 6-cm. cuff. The systolic readings were estimated by the palpation method, the diastolic readings by the change in the character of the pulse-wave to the palpating finger. She admits that the cuff used was too narrow for accurate results. A. Katzenberger[8] made a series of 241 observations on children ranging from 4 to 14 years of age, using a mercurial instrument with an arm-band 9.5 cm. wide. All readings were taken by the auscultation method. To indicate the diastolic pressure she used the time of disappearance of all sound. She found a gradual increase in blood-pressure from 1 to 14 years, and then a sudden rise.

The most recent work of a scientific nature on blood-pressure in children is that of Mello Leitao.[9] His observations were made on healthy infants and children up to 6 years of age. He used an Erlanger apparatus with a 6-cm. cuff, applied to the thigh and the pulse was felt in the posterior tibial artery. The use of the narrow cuff gives a considerable error, as the width of the cuff should be proportional to the circumference of the extremity used. His figures represent the most careful work on blood-pressure in infants.

In reviewing the work of previous investigators it is necessary to determine the value and accuracy of the various methods employed. The palpation method is the oldest one. It gives a fairly correct systolic pressure, but does not give accurately the diastolic pressure. The personal equation in estimating the diastolic pressure has been found so great that this method has been entirely discarded by careful observers. Otfried Müller is authority for the statement that the method of measuring the diastolic pressure by taking the time of lessened pulse-wave (as noted by palpation), is liable to an error of 28 per cent., when compared with readings taken with a manometer tube in an open artery.[10] Since the blood-pressure is lower in children it is much harder to determine the time of appearance or disappearance of the pulse-wave than in adults. We feel that the observations taken by this method cannot be considered accurate.

The oscillatory method, as exemplified by the von Recklinghausen, Pachon and Erlanger apparatus, represents a step in advance. Neither the von Recklinghausen nor the Pachon instruments are recording. As they depend on the spring capsule principle they are very unreliable and require constant standardization; therefore, they will not be further considered. The Erlanger apparatus, however, is a recording instrument and not subject to inaccuracy due to usage. At first sight this apparatus seems to afford an accurate means of blood-pressure

8. Katzenberger, A.: Ztschr. f. Kinderh., 1913, ix, 167.
9. Leitao, Mello: Arch. d. méd. d. enf., February, 1913.
10. Müller, Otfried: Ztschr. f. Kinderh., 1913, ix, 167.

determination in children, but the use of the instrument as devised by Erlanger is difficult and the results are open to question. When the systolic pressure is to be determined, the pressure in the apparatus is raised considerably above the obliteration of the radial pulse, the kymograph drum is started to rotate and the pressure in the cuff is slowly released by means of an ingenious valve. The operator must observe the needle on the drum and the height of the mercury column. At the time there is an abrupt increase in the oscillation of the needle, the observer reads the height of the mercury column and records it as the systolic pressure. Many observers have found it difficult to obtain accurately the systolic pressure by this method. When this instrument is used for the estimation of blood-pressure in children the oscillations are much smaller and the difference in the height of the oscillations is much more difficult to determine. The observation of the diastolic pressure by the continual lowering of the mercury column is open to the same objection, whereas if one releases the pressure in 5-mm. steps, as advocated by Dr. Erlanger, it takes so long for the observation that the nervousness of the child vitiates the results.

The auscultation method was introduced by Korotkoff[11] in 1906 and has replaced the older palpation method in adult work. It is very simple and requires no complicated apparatus. By reading the height of the mercury column at the time the first clear sound is heard (first phase), the correct systolic pressure is obtained. That this method gives accurately the systolic pressure in adults is universally recognized. In children it is not always possible to hear the sounds plainly but in the large majority of cases it can be done. Care should be taken to place the stethoscope at least 1 inch (2 cm.) below the edge of the constricting cuff. As the artery under the cuff is forced against the bone out of its normal relations, it takes at least a distance of 2 cm., or 1 inch below the cuff, before the artery returns again to its normal superficial relations so that we can get the best auscultatory results. The arm should be used whenever possible, as the artery is superficial and auscultation easy. The diastolic pressure can also be accurately determined by this method. After obtaining the systolic reading as described above, one continues to listen over the artery and soon hears a murmur (second phase), replacing the first clear thumping sound. This murmur in turn is replaced by a second tapping sound (third phase) which later becomes duller (fourth phase). Shortly thereafter all sounds disappear (fifth phase).

11. Korotkoff: Tr. Imp. Acad. Med., St. Petersburg, 1905, xv, 365.

Most of the more recent observers hold the view that the beginning of the fourth phase (or dull sound) denotes the time of the true diastolic pressure. The height of the mercury column at this time gives the correct reading. Schrumpf and Zabel[12] made twelve hundred observations of blood-pressure. In the majority of cases the results obtained by the auscultatory method corresponded to the results of the oscillatory method. Lang and Manswetowa[13] determined that the diastolic pressure corresponds to the beginning of the fourth phase. Fischer holds the same view, as does also B. Zabel,[14] who took the beginning of the fourth phase as synchronous with the correct diastolic pressure. Lately, Warfield,[15] Taussig and Cook,[16] have con-

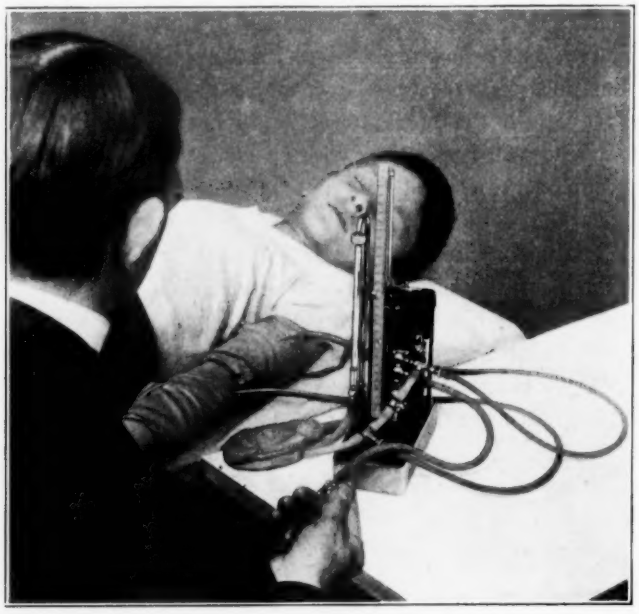

Fig. 1.—Nicholson apparatus with pith-ball attachment.

firmed these observations. Warfield conducted a series of animal experiments which proved this point. The authors are in complete accord with these views and have corroborated them in their work.

12. Schrumpf and Zabel: München. med. Wchnschr., 1909, lvi, 704.
13. Lang and Manswetowa: Deutsch. Arch. f. klin. Med., 1908, xciv, 441.
14. Zabel, B.: Berl. klin. Wchnschr., 1909, xlvi, 1352.
15. Warfield, Louis M.: Studies in Auscultatory Blood-Pressure Phenomena. The Experimental Determination of Diastolic Pressure, Arch. Int. Med., September, 1912, p. 258; Studies in Auscultatory Blood-Pressure Phenomena — the Chemical Determination of Diastolic Pressure, The Journal A. M. A., Oct. 4, 1913, p. 1254.
16. Taussig, Albert E., and Cook, Jerome E.: The Determination of Diastolic Pressure in Aortic Regurgitation, Arch. Int. Med., May, 1913, p. 542.

The pith-ball apparatus was originated by Dr. Fedde[17] as a means of determining diastolic pressure. It was later modified by Dr. Hoobler,[18] to take both systolic and diastolic readings. This instrument was still later modified by one of us. Figure 1 shows the modified apparatus as used by us. The principle utilized in this apparatus is that of oscillation, and rests on the proven work of Sahli.

No matter what method is employed there are certain details as to the size of the cuff, etc., that are essential. Von Recklinghausen[19] has shown the importance of having a cuff between 12 and 15 cm. in width for use on adults, and some investigators have emphasized the importance of the wide cuff in observations on children. With

Fig. 2.—Dr. Nicholson's modification of Erlanger apparatus.

this in mind we have taken readings with cuffs of different width under similar conditions in order to determine how great a significance is to be attached to the width of the cuff. Another detail of technic is emphasized by Schrumpf and Zabel,[12] namely, that the arm-band must be applied snugly and in a completely deflated condition (especially between readings) to avoid causing congestion of the arm. The cuff must not press on the artery at the bend of the elbow. It is also important in applying the cuff to be sure that the

17. Fedde: Med. Rec., New York, July 10, 1910.
18. Hoobler: Med. Rec., New York, Dec. 30, 1911.
19. Von Recklinghausen: Arch. f. exper. Path. u. Pharmakol., 1901, xlvi, 78.

pneumatic pad is placed directly over the line of the artery. All observations must be taken as rapidly as possible, not only to avoid congestion of the extremity but also to prevent the undue excitement incident to prolonged pressure. The most opportune time is between meals, preferably in the afternoon and in the reclining position.

In the progress of our investigations it seemed advantageous to control our readings by another method. Accordingly, Dr. Nicholson modified the Erlanger apparatus in the belief that the personal equation could be practically eliminated. The modification of the Erlanger apparatus is well shown in Figure 2. The ordinary manometer tube is replaced by a larger U-tube having a double scale for reading the height of the mercury column. Resting on the left mercury column is an ivory float, which by an extension rod of aluminum works up

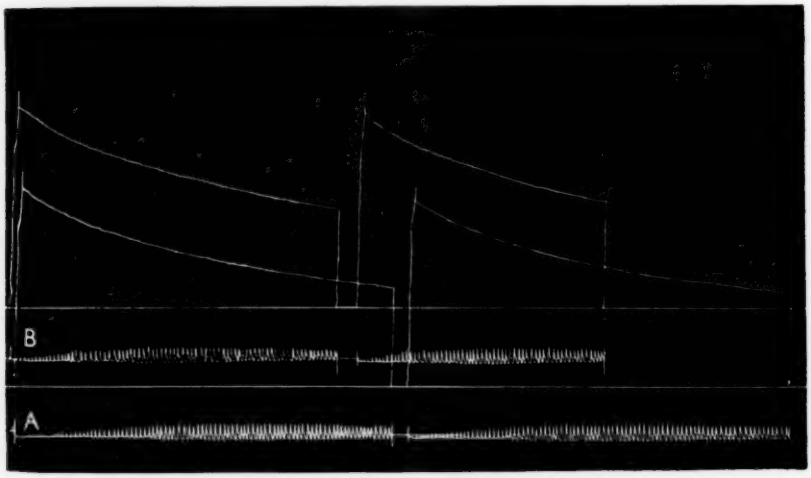

Fig. 3.—Graphic record made with Nicholson modification of Erlanger apparatus. *A*, record made with 13 cm. cuff; *B*, record made with 9 cm. cuff.

and down in a metal trough with a guide ring. At right angles to this extension is fastened an arm carrying a metal movable and adjustable needle, which comes in contact with the smoked paper of the revolving drum. In place of the glass bulb ordinarily supplied with the Erlanger apparatus, a much smaller bulb was substituted. This gives higher oscillations, as it reduces the amount of residual air between the rubber bulb and the interior of the glass bulb. The operation of the modified Erlanger apparatus is very simple. The recording portion of the instrument consists of two movable arms, one directly connected with the mercury float, which indicates on the drum the height of the mercury column. The other arm is activated by a rubber diaphragm, as in the original Erlanger apparatus, and

marks on the drum the oscillation of the blood in the artery under observation. The recording points of the movable arms are so adjusted that they are in the same vertical line. The arm attached to the mercury float is placed in contact with the smoked drum. The drum is then rotated one complete turn and thus indicates by a base-line the zero point of the mercury column. The lower arm is swung into position against the drum, and the apparatus operated as in the original Erlanger. The upper indicator records the height of the mercury column and the lower one shows the pulsation of the artery. The pressure is allowed to fall and the drum kept revolving, until the oscillations of the lower indicator have reached and passed their maxima, and are permanently lower. A glance at the record will help to make clear the operation of the modified apparatus (Fig. 3). The interpretation of the record is made as suggested by Erlanger. The point where the first abrupt increase in the size of the oscillations

TABLE 2.—PULSE-PRESSURE IN CHILDREN, VARIOUS OBSERVERS

Observers	Years												
	3	4	5	6	7	8	9	10	11	12	13	14	15
M. Leitão	26.0	28.0	28.0	....	....	....	....	....	....	....	....	....	....
W. Kriss	6.0	7.0	7.0	8.0	8.0	2.0	2.0	8.0	8.0	4.0	4.0	5.0	5.0
W. Kaupe	8.2	8.1	8.2	15.4	13.0	10.3	8.5	15.1	8.8	8.9	8.6	11.0	....
A. Katzenberger.	....	39.0	40.5	29.0	....	48.0	42.0	34.1	21.0	40.0	37.5	48.0	....
Judson-Nicholson	26.2	26.7	26.9	25.3	26.9	28.9	23.3	32.1	31.5	37.1	33.1	38.7	38.1

occurs, or where there is a characteristic drop in the base-line, is taken as the systolic pressure. The figures in millimeters of mercury are obtained by applying a millimeter rule at right angles to the lower curve, and measuring the distance from the point where the rule bisects the base line of the upper curve to the curve proper, then doubling the figures obtained. The doubling of the figures is necessary to allow for the depression of one arm of the mercury column in the U-tube, while the other is being elevated. The diastolic pressure is obtained by determining the point where the pulsations begin to diminish in height, and by measuring from the base-line to the corresponding point in the upper curve. In certain cases the oscillatory curve reaches its maximum height, then gradually diminishes, only to rise again to a second maximum of oscillation, following which is a gradual and continuous lowering of the oscillations.

We have followed Dr. Erlanger's interpretation and used the second maximum oscillation as designating the correct diastolic pres-

sure. This method gives a permanent record which is rapidly and accurately taken and eliminates the long time necessary, when the intermittent method of determining the diastolic pressure is used (as recommended by Dr. Erlanger).

In conducting our investigations we have used readings taken by three main methods, auscultation, oscillation with the pith-ball apparatus and the modified Erlanger apparatus. All readings were taken between meals with the children in a reclining position, and the children rested between readings. In all cases we used the largest cuff available in proportion to the arm and a smaller cuff for comparative results. The children ranged from 3 to 15 years and were in daily

TABLE 3.—BLOOD-PRESSURE—

Observers	Method	Cuff cm.	Age, Years									
			3		4		5		6		7	
			Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.
L. Gordon	Palpation	12	81.0	.....	83.0	.....	86.5	.....	88.5	.....	85.0	.....
Stowell	Palpation	12	91.0	.....	89.0	.....	95.0	.....	96.0	.....	102.0	.....
Leitão	Erlanger	6	91.0	65.0	99.0	71.0	99.0	71.0	.....	.....	.....	.....
Kaupe	Palpation	12	90.0	81.8	86.4	78.3	82.2	74.0	97.5	82.1	91.0	78.0
W. Kriss	Auscultation Dias. 5 Phas.	5	80.0	74.0	83.0	76.0	83.0	76.0	90.0	82.0	90.0	82.0
Katzenberger	Auscultation Dias. 5 Phas.	9.5	....	.....	93.0	54.0	94.5	54.0	82.0	53.0	.....	.....
Judson and Nicholson	Modified Erlanger	13 and 9	91.8	65.6	91.6	64.9	91.3	64.4	92.6	67.3	94.0	66.3

attendance at the public schools, residing at the Foster Home and the Southern Home. Most of these children were well nourished and in good physical condition, and a number of them were above the average in development. After school hours, they were occupied with outdoor sports and games, for which there were ample facilities. We consider them representative of the average child.

Our studies started one year and a half ago and cover some two thousand three hundred observations. In a large number of cases we took readings by the auscultation method, while the records were being made on the drum of the modified Erlanger apparatus, so that they afford a very good means of comparison of the two methods. We believe that we have established a standard of blood-pressure in the normal child between the ages of 3 and 15 years. The results obtained with the modified Erlanger apparatus we have taken as the standard.

That they are correct becomes apparent when we consider the uniformity of the readings for the systolic and diastolic pressures. Comparison of the results obtained by the conjoint use of the auscultation and modified Erlanger methods further confirms their accuracy. The method of recording the results on the modified Erlanger apparatus thoroughly eliminates the personal equation, as both the oscillatory curve produced by the pulse-wave and the height of the mercury column are recorded automatically on the moving drum. The finished records can be studied and accurately interpreted at our leisure, determination of the systolic and diastolic pressure being easily obtained by careful measurements of the curve.

—IN NORMAL CHILDREN

Age, Years															
8		9		10		11		12		13		14		15	
Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.	Sys.	Dias.
93.0	.....	100.0	.....	95.0	.....	104.0	.....	105.0	.....	.....	.....	.....	.....	.....	.....
101.0	.....	102.0	.....	112.0	.....	102.0	.....	111.0	.....	107.0	.....	110.0	.....	109.0	.....
.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
90.5	80.2	90.0	81.5	97.7	82.6	96.0	87.2	92.0	83.1	96.6	88.0	107.0	96.0	.....	.....
90.	88.0	90.0	88.0	98.0	90.0	98.0	90.0	99.0	95.0	99.0	95.0	101.0	96.0	101.0	96.0
103.0	55.0	100.6	58.6	104.6	69.5	99.8	78.8	108.2	68.2	107.7	70.2	119.2	71.2	.....	.....
93.6	64.7	94.3	71.0	99.2	67.1	97.1	65.5	102.3	65.2	103.6	70.5	106.1	67.4	105.6	67.5

The criticism may be made that, while the table represents the summary of a large number of observations, the individual variations might still be so considerable as to impair its value. We find that the widest variations occur from the tenth to the fourteenth year when there is a marked rise in the systolic pressure. From the fourth to the fourteenth year, inclusive, the variations do not exceed 5 mm. in two-thirds of our cases, and in three-fourths of them, the blood-pressure shows a variation of less than 8 mm.

The systolic pressure shows a slight but gradual rise from 3 to 10 years. From 10 to 14 years the increase is more abrupt, with a rapid elevation in the fourteenth year during adolescence. The systolic pressure varies from 91 mm. in the fourth year to 105.5 mm. in the fourteenth year.

Our results show that the systolic pressure is higher than that generally accepted and does not show any arithmetical increase from year to year. The total rise in systolic pressure from 4 to 14 years is represented by only 14 mm. of mercury.

In contrast to the systolic pressure, the diastolic remains at an almost uniform level, and the pulse-pressure increases progressively and proportionately more than the systolic pressure over the corresponding period. Comparing the systolic pressure at 4 years with that of 14 years, we find an increase of 16 per cent., whereas the increase in the pulse-pressure during the same period amounts to 45 per cent.

Comparing the results obtained by using cuffs of different width, we find that from 4 to 8 years the circumference of the arm varies from 13 to 18 cm., and that the 9-cm. cuff gives as accurate results as when wider ones are used (11 to 13 cm.). From 8 to 15 years the circumference of the arm varies from 14 to 25 cm. During this period we find more accurate results when using the 13-cm. cuff. Concerning the influence of the pulse rate, we believe that the large number of observations taken on the same child practically eliminates errors due to this cause.

Comparison of the results obtained by the auscultation method with those of the modified Erlanger method, show remarkable uniformity in the readings. The auscultation method requires no complicated apparatus, is easily carried out, and gives accurate results if the necessary precautions are taken to observe correct technic, especially as to the mode of application and width of the cuff.

In comparing the results of the auscultation with other methods, we find the average variation in the systolic pressure ranged from 1 mm. more to 6.5 mm. less than the readings obtained when using the modified Erlanger apparatus.

The diastolic readings on the other hand, were from 1 to 5 mm. lower, except at 9 years and 13 years when they were respectively 9 and 7 mm. lower, the result is a higher pulse-pressure averaging 4 mm. for the period covered in our tables.

On reviewing the results obtained by the pith-ball method, we find the systolic pressure ranges from 7 mm. above to 1 mm. below the readings of the modified Erlanger. The diastolic pressure varies from 2 mm. above to 5 mm. below, with the exception of the eighth year, when it was 8 mm. above the figure on the modified Erlanger instrument. This produces an average increase of 1.5 mm. in the pulse-pressure.

When we use the auscultation method in children, we must take the time of appearance of the dull sound (fourth phase) as the time

to read the height of the mercury column, which gives the correct diastolic pressure. If the time of disappearance of all sound (fifth phase) is taken, the error varies from 11 to 20 mm. This makes a very marked difference in the pulse-pressure.

The results obtained with the pith-ball apparatus compare favorably with those obtained by the other two methods. In our table the figures given for children aged 3 years and 15 years, were not utilized in drawing conclusions, as the number of observations was too small.

In closing we must insist on the importance of repeated observations in a given case before reaching conclusions. The figures in our tables represent the averages of a large number of readings; we do not claim that they are an *absolute* standard. While in the individual child the variations in the systolic pressure may be considerable, diastolic pressure remains fairly constant. The importance of determining the diastolic pressure in every case cannot be too strongly emphasized. The determination of the pulse-pressure, indicating as it does the peripheral resistance, is the most important point to be determined in children.

A STUDY OF THE BLOOD-PRESSURE IN ANEMIA IN INFANCY*

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This study was undertaken because of the unexpected results obtained in an examination of the blood-pressure in a marked case of anemia in an infant. The history of this case will be given in detail further on. It was found on looking up the literature that there were very few data as to the blood-pressure in infancy, whether in health or in disease, and practically nothing as to the blood-pressure in anemia in infancy.

Shaw,[1] using Gärtner's tonometer, made 400 measurements on forty-five children ranging in age from 3 months to 12 years. He found that the systolic pressure under normal conditions varied between 90 and 110 mm. He also found that the age of the child appeared to have very little influence on the height of the blood-pressure.

Cook and Briggs[2] found that the systolic pressure during the first two years varied between 75 and 90 mm. They used a modification of the Riva-Rocci instrument and determined the pressure by palpation. They do not state the width of the band used, and give no data as to the number of babies examined.

Beretta[3] found that the systolic pressure in babies during the first year varied between 54 and 87 mm., the average being 76 mm. He used the Riva-Rocci instrument.

Stone[4] found that the ordinary reading in infants under 6 months was between 60 and 70 mm., and between 2 and 3 years, 80 to 90 mm. He used the Riva-Rocci apparatus, but gives no data as to

*From the Medical Service of the Children's Hospital, Boston.
*Read before the Section on Diseases of Children at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

1. Shaw: Albany Med. Ann., 1900, xxi, 88.
2. Cook and Briggs: Johns Hopkins Hosp. Rep., 1903, xi, 451.
3. Beretta: Abstr. in Monatschr. f. Kinderheilk., 1903-4, ii, 725.
4. Stone: Boston Med. and Surg. Jour., 1904, cl, 262.

the width of the cuff used, where the pressure was taken or how many babies were examined.

Oppenheimer and Bauchwitz[5] examined sixty-eight children ranging from the new-born to 14 years of age. They do not give the number of children tested at the different ages. They used a modification of the Riva-Rocci instrument, with a cuff 5 cm. in width. They state that the blood-pressure between birth and 6 months is 80 mm., from 7 months to 1 year, 90 mm., and from 2 years to 3 years, 90 mm.

Trumpp[6] made 1,062 observations on fifty-six infants, using Gärtner's tonometer. He found that the systolic pressure in healthy, quiet infants varied between 60 and 90 mm., the average being 80 mm. Readings between 60 and 70 mm. were ordinarily found only during sleep or in premature or feeble infants. The pressure was the same in the new-born as in older infants. Nervous excitement and muscular activity increased the pressure from 10 to 60 mm. The taking of food increased it from 8 to 10 mm. He studied nine cases of acute and eight of chronic disturbances of nutrition and found that the blood-pressure sank with the increase in the debility. Anything under 50 mm. was a bad sign. When there were marked changes in weight, the blood-pressure rose and fell with the weight. The blood-pressure rose with water retention.

Gordon[7] studied 170 normal children, but does not state the number of children at the various ages. He used Martin's modification of the Riva-Rocci instrument, with a cuff 12 cm. in width. He found that the systolic pressure under 1 year was 71 mm., at 1 year, 73 mm., and at 2 years 79.5 mm. He states that the figures are higher if a narrower armlet is used.

Hill[8] made nineteen examinations on sixteen babies under 2 years of age, ill with gastro-enteritis. The average systolic pressure in these cases was 102 mm., the average diastolic, 81 mm., and the average pulse-pressure, 21 mm. The highest systolic pressure was 130, the lowest, 85; the highest diastolic, 118, and the lowest, 60; the highest pulse-pressure 36 and the lowest 8. He emphasizes the importance of the pulse-pressure, and says that it is far more important than the systolic pressure alone. The pulse-pressure was somewhat lower in the infants that died, but there were many exceptions. He concludes that the blood-pressure is not of the same importance in infants as in adults. He used the Nicholson sphygmomanometer, a modification of the Riva-Rocci type. The instrument was applied on the

5. Oppenheimer and Bauchwitz: Arch. f. Kinderheilk., 1905, xlii, 415.
6. Trumpp: Jahrb. f. Kinderh., 1906, lxiii, 43.
7. Gordon: Arch. Pediat., 1911, xxviii, 343.
8. Hill: Arch. Pediat., 1913, xxx, 588.

TABLE 1.—Normal Infants

No.	Age in Months	Sex*	Systolic Pressure	Diastolic Pressure	Pulse Pressure	Hemoglobin %
1	2	♀	80-76	50-57	30-19	
2	2¼	♀	72	58	14	70
3	[illegible]	♂	84	62	22	
4	3	♂	78	65	23	
5	3	♀	100-97	70-74	30-23	
6	3	♂	78	64	14	
7	3	♂	78	61	17	
8	4	♂	82	69	13	
9	4	♂	88-90	52-57	36-33	75
10	4½	♂	80-92	44-60	36-32	80
11	5	♂	82	64	18	
12	6	♂	92-96	75-74	17-22	
13	6	♂	98	82	16	
14	6	♂	88	72	16	
15	6	♀	94-95	40-60	54-35	
16	6	♀	86	67	19	
17	6	♂	84-87	44-56	40-31	80
18	6½	♂	98	78	20	80
19	7	♀	90-92	50-57	40-35	
20	7	♀	90	76	14	
21	8	♀	86	63	23	
22	8	♂	83	74	9	
23	8	♀	95	64	31	
24	8	♂	80-94	40-52	40-42	
25	8	♀	110	80	30	
26	8	♂	88	59	29	
27	8½	♂	80-92	45-56	35-36	
28	8½	♂	95	70	25	80
29	9	♀	87	68	19	
30	9	♂	110	80	30	
31	10	♂	102-100	76-80	26-20	70
32	10	♂	87	72	15	
33	11	♂	85	60	25	
34	11	♀	82	69	13	
35	11	♀	82	59	23	
36	11	♂	89	66	23	
37	11	♀	92	78	14	
38	11	♀	100	76	24	
39	12	♂	94-92	70-73	24-19	80
40	12	♂	100	74	26	
41	12	♀	95	65	30	
42	12	♂	90	75	15	
43	12	♀	92	60	32	
44	13	♂	98	76	22	
45	13	♀	92	60	32	
46	14	♂	100	80	20	
47	14	♂	97	83	14	
48	15	♂	100	79	21	
49	17	♂	100	70	30	
50	22	♂	104	84	20	
Average			90	66	25	

*In this and the following tables ♂ stands for male and ♀ for female.

thigh and the readings made by auscultation over the popliteal artery. The systolic pressure was measured from the first sound heard, and the point at which the sound entirely disappeared was taken as the diastolic pressure.

It seemed advisable, therefore, to examine first a series of normal infants, in order to get a more definite normal standard. A series of poorly nourished babies, not suffering from anemia, was then examined to determine what effect, if any, the condition of the nutrition had on the blood-pressure. A small series of babies suffering from moderate anemia was then examined, and finally, a more detailed examination of the baby suffering from severe anemia was made.

The "Tycos" sphygmomanometer was used in this study. The width of the band was 5 cm. The tests were made on the arm, when possible, otherwise on the thigh. As a matter of fact, about one-half of them were made on the arm and one-half on the thigh. The determinations of the blood-pressure were all made with the stethoscope, the bell being applied over the vessels at the elbow when the cuff was applied on the upper arm, and in the popliteal space, when it was applied on the thigh. The systolic pressure was read when the sound was first heard; the diastolic pressure was read when the sound changed from sharp to dull.

Sixty-two observations were made on fifty normal babies. These results are given in detail in Table 1. The average systolic pressure in normal babies under 2 years of age was, therefore, 90 mm., the average diastolic, 66 mm., and the average pulse-pressure 25 mm.

TABLE 2.—COMPARISON BETWEEN VARIOUS PRESSURES IN THE FIRST AND SECOND YEARS

	First Year	Second Year
Number of Cases	38	12
Average Systolic Pressure	89	95
Average Diastolic Pressure	64	73
Average Pulse-Pressure	25	24
Highest Systolic Pressure	110	104
Lowest Systolic Pressure	72	90
Highest Diastolic Pressure	82	84
Lowest Diastolic Pressure	40	60
Highest Pulse-Pressure	54	32
Lowest Pulse-Pressure	9	14

Thirty-two of these babies were males and eighteen females. The average systolic pressure in the males was 91 mm. and in the females 86 mm. The average diastolic pressure in the males was 68 mm. and in the females, 64 mm. The average pulse-pressure in the males was

TABLE 3.—Poorly Nourished Infants

No.	Age in Months	Sex	Systolic Pressure	Diastolic Pressure	Pulse-Pressure	Hemoglobin Red Corpuscles	Diagnosis	General Condition	Spleen
1	1½	♂	74	36	38	80%	Indigestion, malnutrition	Poor	
2	2	♂	74	45	29		Pyloric spasm	Poor	
3	2	♂	69	40	29		Pyloric spasm	Poor	
4	2	♀	72	43	29		Feeding	Poor	
5	2½	♀	64	40	24	70%	Malnutrition	Fair	Spleen 2 cm.
6	3	♀	72	46	26		Malnutrition	Poor	
7	3	♂	60	47	13		Pyloric spasm	Poor	
8	3	♀	75	40	35		Indigestion	Poor	
9	4	♀	80	50	30		Feeding	Good	
10	4	♀	84	57	27		Pyloric spasm	Poor	
11	5	♂	80	40	40		Feeding	Fair	
12	5	♂	93	54	39		Indigestion	Fair	
13	5	♀	60	40	20		Malnutrition	Poor	
14	5	♂	83	52	31		Malnutrition	Poor	
15	7	♂	70	30	40	80%	Rachitis	Fair	Spleen +
			75	30	45	4,000,000			
16	7	♂	92	35	57	70%	Malnutrition	Good	Spleen +
			90	40	50	4,550,000			
17	7	♀	94	63	31		Feeding	Poor	
18	10	♂	92	51	41		Syphilis	Poor	
19	10	♀	90	45	45		Scorbutus	Fair	
20	11	♀	87	60	27		Pyloric spasm	Poor	
21	11	♂	100	65	35		Malnutrition	Fair	
22	12	♂	90	72	18		Malnutrition	Fair	
23	13	♂	106	68	38		Malnutrition	Poor	
24	15	♂	102	73	29	80%	Otitis media	Poor	
						60%			
25	19	♂	87	60	27	6,480,000	Rachitis	Fair	
Average	.....	.....	89	53	36				

25 mm. and in the females, 27 mm. These differences are so slight that they are presumably unimportant. They do show, however, a slightly higher pressure, in both systole and diastole, in the males than in the females.

The highest systolic pressure was 110, and the lowest 72 mm., while the highest diastolic was 84, and the lowest diastolic 40 mm. The pulse pressure varied between 54 mm. and 9 mm.

TABLE 4.—ANEMIC INFANTS

Number	Red Corpuscles	Hb., %	Spleen	Age in Months	Sex	Systolic Pressure	Diastolic Pressure	Pulse-Pressure	Murmur in Neck	Murmur in Heart	Murmur in Groin	Pistol Shot
1	2,000,000	40	Palpable at navel	4	♂	114	42	72	Systolic	Systolic at base	0	0
2	2,400,000		0	12	♂	120	40	80	Systolic	Systolic over whole precardia	Systolic	0
	2,600,000	30	.....	....	♂	95	60	35	Systolic	Systolic over whole precardia	Systolic	0
	2,640,000	31	.....	...	♂	120	55	65	Systolic	Systolic over whole precardia	Systolic	0
3	3,000,000	60	2 cm.	4	♂	100	20	80	Systolic	Systolic	0	0
4	3,200,000	70	Enlarged	8	♀	100	45	55	Systolic	Systolic	0	0
						105	45	60				
5	3,496,000	65	Just Palpable	2	♂	60	25	35	0	0	0	0
						65	29	36				
6	3,500,000	70	0	3½	♂	90	40	50	Systolic	0	0	0
7	3,744,000	65	0	10	♂	96	40	56	0	0	Systolic	0
8	3,800,000	80	0	4	♂	84	48	36	0	0	0	0
9	3,800,000	75	0	8	♂	105	53	52	Systolic	0	0	0
10	4,000,000	50	0	8	♂	100	35	65	0	0	Systolic	0
						110	35	75				
	Average		.....			97	41	57				

Table 2 gives a comparison between the various pressures in the first year and in the second year. These figures seem to show that both the systolic and the diastolic pressure are somewhat higher during the second than during the first year, while the pulse-pressure is the same. The number of cases is too small, however, to warrant very definite conclusions on these points.

Twenty-seven observations were made on twenty-five poorly nourished, but not anemic, babies. These results are given in detail in Table 3.

The average systolic pressure in these babies was, therefore, 89 mm., the average diastolic, 53 mm., and the average pulse-pressure, 36 mm. The highest systolic pressure was 106 mm., and the lowest 60 mm., while the highest diastolic pressure was 73 mm. and the lowest diastolic pressure 30 mm. The highest pulse-pressure was 57 mm. and the lowest 13 mm. Comparison of the blood-pressures in the normal and poorly nourished babies shows that, while the average systolic pressure was practically the same in both cases, the diastolic pressure was lower and the pulse-pressure, therefore, higher in the poorly nourished babies.

Fifteen observations were made on ten babies suffering from anemia. These cases have been arranged in Table 4 according to the number of red corpuscles and not according to the age. This table shows that the systolic pressure was somewhat higher in these babies than in either the normal or the poorly nourished babies, while

TABLE 5.—

Age in Months	Hb., Per cent.	Red Corpuscles	White Corpuscles	Spleen, cm.	Liver, cm.	Systolic Pressure
12	25	1,800,000	28,000	5	2	124
14	55	2,720,000	47,000	6	4	100
18	70	3,776,000	?	4	4	110
24	?	?	?	13	5	120
25	40	2,480,000	19,500	13	5	124

the diastolic pressure was considerably lower and the pulse pressure, therefore, much higher. There was no definite relation, however, between the amount of the pulse-pressure and the degree of the anemia.

The most interesting observations were those in the baby suffering with severe anemia, to which reference has already been made. This baby was 12 months old when first seen. He was markedly pale, but not badly nourished. The left border of the heart was 6 cm. to the left, and the right border 2.25 cm. to the right of the median line, while the upper border was at the upper border of the third rib. The first sound over the whole precordia was followed by a blowing murmur, loudest in the third left interspace. It was transmitted better to the left axilla than to the right. The second pulmonic was considerably louder than the second aortic sound. There was no thrill. There was a pistol-shot in the groin. There was also a systolic murmur, but no diastolic murmur, in the groin. There was

a Corrigan as well as a capillary pulse. The extremities were not clubbed. The lower border of the liver was 2 cm. below the costal border in the nipple line, while the lower border of the spleen was 5 cm. below the costal border. The hemoglobin, estimated with the Sahli apparatus, was 25 per cent. There were 1,800,000 red corpuscles and 28,000 white corpuscles. The red corpuscles showed marked variation in size and shape, with marked polychromatophilia. There were many stippled cells. Thirteen normoblasts were seen in counting 100 white cells. A differential count showed: mononuclears, 44 per cent.; transitional forms, 12 per cent.; polynuclear neutrophils, 39 per cent.; eosinophils, 5 per cent.

The systolic blood-pressure was 124, and the diastolic pressure 0, giving a pulse-pressure of 124 mm. That is to say, the conditions in the peripheral circulation corresponded exactly to those found in aortic regurgitation in adults. It was thought for a time that there

—Severe Anemia

Diastolic Pressure	Pulse-Pressure	Murmur in Heart	Bruit in Neck	Murmur in Groin	Pistol Shot
0	124	Systolic over whole precordia	Systolic	Systolic	+
15	85	Murmur less marked.	Systolic	Systolic	0
30	80	Very slight systolic at base	Systolic	0	0
0	120	Systolic at base	Systolic	Systolic	+
30	94	Systolic at base	Systolic	0	0

must be an aortic regurgitation. Then the query arose as to whether these conditions might not be due to some change in the peripheral circulation as the result of the severe anemia. Further observations on the baby, which are detailed in Table 5, show that the latter was the true explanation.

When he was last seen, when 25 months old, he was fairly developed and nourished, but still markedly pale. He showed many of the changes in the bones characteristic of rickets. His heart was of normal size. There was a systolic murmur at the base of the heart, loudest in the pulmonic area. It was also audible along the sternum to the fourth left space. There was a bruit in the neck. There was a systolic murmur in the groin, but no pistol-shot. The liver was palpable 5 cm. below the costal border in the nipple line. The spleen filled the whole left half of the abdomen, extending 3 cm. beyond the median line.

The examination of the blood gave the following results:

Hemoglobin	40 per cent. (Sahli)
Red corpuscles	2,480,000
White corpuscles	19,500
Small mononuclears	28 per cent.
Large mononuclears and transition forms	76 per cent.
Polynuclear neutrophils	6 per cent.
Eosinophils	0 per cent.

The red corpuscles showed marked achromia and there was considerable variation in their size and shape, as well as polychromatophilia and basic stippling. Eight normoblasts and one megaloblast were seen while counting 200 white cells.

Comparison of the observations in this instance with those in the other anemic babies, the poorly nourished babies and the normal babies, show that with disturbances of nutrition there is a progressive lowering of the diastolic pressure and a corresponding increase in the pulse pressure. The systolic pressure, on the other hand, rises with the increase of the anemia. These observations merely bring out certain facts but, unfortunately, do not warrant any definite conclusions as to their cause.

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FURTHER ANALYSIS OF THE VOICE SIGN IN CHOREA *

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Elsewhere[1] I have reported in a short preliminary note on the voice in chorea as analyzed by the aid of the vocal kymograph. This was merely the first blush, the original snap-shot of a single isolated case.

That note briefly reviewed the literature since 1841, showed the vagueness of former descriptions of choreic vocal changes, the antiquated methods of vocal analysis, the advantages of modern methods, and finally presented — as far as I know — the first clear statement of just what choreic voice is. Without reiterating at length the details of that preliminary note, let me briefly state that the voice-change as reported on the kymograph consists in a variation in two vocal elements — a rise in pitch and an increase in intensity. This vocal change was so constant and so uniformly simultaneous with other choreiform movements that I there presented the claim that these vocal changes deserved at least to be classed with Shaw's[2] knee-jerk in chorea and with Graves'[3] respiratory signs in chorea, and should therefore in routine clinical examination be called the voice sign in chorea.

From the start I felt that the preliminary note should be followed up by a variety of tests carried uniformly through a long series of cases in order to ascertain the varieties of the voice sign in different aspects of the disease — early, marked, late, recovered, partial and hemichorea — as well as to ascertain if change in pitch and vocal intensity were the only effects of choreic movements on vocal utterance; also the varieties of these in different stages and forms if any.

It is the procedures and results of this research that I now present. Let me here express my appreciation to Prof. John J. Thomas for opportunity to carry on this investigation.

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* Read before the Section on Nervous and Mental Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

* From the Neurological Department of Boston City Hospital, Service of Prof. John Jenks Thomas.

1. Swift, Walter B.: A Voice Sign in Chorea: Preliminary Note, Am. Jour. Dis. Child., June, 1914, p. 422.

2. Shaw, H. L. K.: The Knee-Jerk in Chorea, Albany Med. Ann., May, 1897.

3. Graves, W. W.: A Study of the Respiratory Signs of Chorea Minor, The Journal A. M. A., Jan. 30, 1909, p. 364.

In conversation, vocal changes are short in duration, and hence difficult of perception. This suggested the prolongation of tests to make vocal change more easily perceived. The change recorded from prolonged tests must necessarily be the same changes elongated that occur in conversation, where the sounds are short.

Twenty cases were then subjected to the following series of twenty-seven tests, 540 tests in all:

1. Prolonged utterance of a as in arm.
2. Prolonged utterance of e as in eye.
3. Prolonged utterance of i as in ice.
4. Prolonged utterance of o as in old.
5. Prolonged utterance of u as in rude.
6. Prolonged utterance of a as in ask.
7. Prolonged utterance or e as in end.
8. Prolonged utterance of i as in ill.
9. Prolonged utterance of o as in odd.
10. Prolonged utterance of u as in up.
11. Consonant p.
12. Consonant b.
13. Consonant m.
14. Consonant t.
15. Consonant d.
16. Consonant n.
17. Consonant k.
18. Consonant g.
19. Consonant r.
20. Consonant l.
21. Consonant s.
22. Prolonged whisper.
23. Prolonged whistle.
24. Blowing the breath.
25. Holding the breath in inspiration.
26. Holding the breath in expiration.
27. Holding the breath in half expiration.

The purpose in trying so many tests was to expose all the forms and modes of vocal production to the choreic twitch, and in this way ascertain the mode and form acted on with highest frequence to serve as a single test adapted to routine clinical examination.

SYMPTOM FREQUENCY IN TWENTY CASES

After tabulating 20 cases of supposed chorea, 4 were found recovered, and 1 a question of hysteria. Excluding those 5, 15 are left, of certain diagnosis on which to base results.

In general, variation in pitch and intensity occurred in two-thirds of the cases pretty uniformly distributed over all the vowel sounds, long and short, with a slightly more frequent change in pitch than intensity. Therefore I place pitch first in mentioning them. There is, however, one marked difference in all these vowel changes. Long "a" is more marked in its change. This is a good reason for its choice

as the routine clinical test. The explanation may lie in the open position of vocal agents in the utterance of "a" long, as in "are," thus allowing any contraction to show more in its effect, than if exerted on a closed position of those agents as "e" in "end."

The whisper changed in pitch in 3 cases, in intensity in 6, showing periodic cessation in 3. The whistle showed irregularity in 3 on expiration, and in 3 cases on inspiration. One case entirely stopped whistling. Three cases could never whistle. One showed periodic cessation of tones, with no change in pitch or intensity determinable.

Consonants showed no change except when prolonged, where three cases showed "e" and "r" to change in pitch and intensity.

Air blow: 4 cases were irregular; 3 halting.

Air held in inspiration, no change.

Air held in expiration showed one puff.

Air held in half expiration showed one puff.

OTHER SIDE OBSERVATIONS NOTED IN THE 540 TESTS OF TWENTY CASES

Cases recovered in from three to five months showing no voice sign.

Also one case recovered in eight months showed no voice sign.

The frequency of occurrence slightly increased with the increase of fatigue.

Once was observed a nasal puff of air, simultaneous with a choreic twitch, mouth closed.

One relapsed case with motions in limbs only showed no voice sign whatever.

One case hemichorea showed vocal changes, like the others.

One case with a uniform and constant vowel variation in pitch and intensity, and that, too, in all the vowels tested showed no vocal change in prolonged "l" and "r."

Voice changes not always in proportion to the amount of choreic motion as seen.

With air held in inspiration, expiration and half expiration, slight movements showed no change in 8 cases.

Marked timidity and slight questionable voice changes should not be counted as positive change.

Slight lack of articulation control or inattention may contribute slight negligible voice changes.

Marked contractions accompany marked voice change.

CONCLUSIONS FROM TWENTY CASES

1. These choreic voice changes are more frequent in the vowels, less so in whisper, whistle, consonants, air blow and holding of breath; and are less and less in frequence in this order.

2. There is sufficient uniformity and frequency in the appearance of vocal changes to warrant us in classifying changes of pitch and intensity as one of the signs of chorea; of equal dignity with the choreic knee-jerk of Shaw,[2] the respiratory signs of Graves[3] and other minor symptoms.

3. Other less frequent and less marked changes occur that seem of interest subordinate to those in the vowels.

4. The most marked change occurred in the open prolonged sound of "a" as in "are," and I therefore offer this as the routine clinical test and method for the elicitation of the choreic voice sign.

SUMMARY

Analysis of twenty cases of chorea with over five hundred observations on the voice show a change of pitch and intensity in two cases out of three — a change that deserves recognition as a new sign in chorea — the choreic voice sign.

110 Bay State Road.

THE DIAGNOSIS AND TREATMENT OF "LATE" HEREDITARY SYPHILIS *

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RELATION OF "EARLY" TO "LATE" SYPHILIS

During the past two years we have seen 123 cases of manifest hereditary syphilis at the St. Louis Children's Hospital. Of these seventy-four, or 60 per cent., belonged to the group commonly described as "late" hereditary syphilis while only forty-nine, or 40 per cent., were infants under 1 year of age with the classical symptoms of rash, coryza, and enlarged spleen which characterize the "early" type. The division into "early" and "late" is arbitrary and only a matter of convenience, as these "early" symptoms may first develop after infancy, while "late" lesions may develop early in infancy. The "late" lesions may also develop primarily in older children without any "early" lesion having occurred or been recognized. Thus lesions comparable with the secondary (eruptive), tertiary (gummatous), and quarternary or para-syphilitic (vascular and nervous lesions), stages of acquired syphilis, are included among the clinical manifestations of "late" hereditary lues.

In only twelve of our cases were we able to obtain a positive history of an "early" lesion in infancy, while in twenty-five cases the history was definitely negative in regard to this point. In the remainder the history was indefinite or could not be obtained. It is highly probable that some of our forty-nine "early" cases will later develop other symptoms and thus ultimately belong to the group under consideration. In connection with the large group of cases without early lesions it is interesting to note a study[1] of the Wassermann reaction in 101 infants in St. Louis (thirty-three of whom were from this clinic) who showed no clinical evidences of syphilis. But two positive and one doubtful reactions were obtained. Although this series is small, it would tend to show that the latent infections in infancy are not so extremely common in St. Louis as might be imagined from our figures.

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* From the Department of Pediatrics, Washington University Medical School and the St. Louis Children's Hospital.

* Read at the annual meeting of the American Pediatric Society, Stockbridge, Mass., May 28, 1914.

1. Blackfan, Nicholson and White: AM. JOUR. DIS. CHILD., 1913, vi, 162.

We are now making a study of latent infection in older children by testing the blood and examining the brothers and sisters of the patients in our series. A large percentage of such children have given positive Wassermann reactions although they have never shown clinical evidences of luetic infection. Many items of interest have occurred in these familial studies which will be reported later.

GENERAL STATISTICS

The ages of the seventy-four patients were as follows:

Years of age	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12
No. of cases	6	4	6	4	6	4	10	7	5	5	11
Years of age	12-13	13-14	14								
No. of cases	2	2	2								

Eighty-three per cent. of the patients were white and 17 per cent. colored, while of the total clinic attendance 92 per cent. are white and 8 per cent. colored. Thirty-three were males and forty-one were females.

We were able to obtain a definite history of the feeding in infancy in twenty-seven cases. Twenty-three had been breast-fed and only four artificially fed. This may be looked on as an illustration of the better chances for life of the breast-fed syphilitic infant over the artificially fed, as the ratio is much different from the ratio of the breast to bottle-fed infants in general.

CLINICAL LESIONS

The following list of clinical diagnoses shows the wide variety of lesions encountered. In many cases there were multiple lesions, and the following figures give the number of cases in which each lesion occurred. In every case there was a positive Wassermann reaction which was checked in practically every instance by a second test.

Bones—	
Periostitis tibia	3
Periostitis skull	1
Joints—	
Acute arthritis knee	7
Acute arthritis ankle	1
Skin—	
Macular eruption	1
Alopecia	2
Condyloma anus	3
Gummata	3
Eye—	
Interstitial keratitis	17*
Choroiditis	1
Central nervous system †—	
Mental deficiency	14
Cerebrospinal syphilis	8
Hemiplegia	5
Epilepsy	4
Paralysis	3
Chorea	2
Hydrocephalus	2
Ulcerations—	
Nasal	2
Laryngeal	1
Pharyngeal	1
Ozena	1
Enlarged spleen (only lesion)	1
Torticollis	1
Aortitis	1
Obscure abdominal pain	1
Obscure pain in legs	2

* 23 per cent.

† Thirty-two patients in all or 43 per cent.

The large number (43 per cent.) of our patients with lesions of the central nervous symptoms is one of the most interesting features from the standpoint of diagnosis. During the past two years we have tested the blood of thirteen patients with hemiplegia admitted to the wards of the hospital of which five reacted positively. In one case—a girl of 5 years—admitted at the onset of the trouble, we were able to limit the extent of the lesion by a vigorous treatment. Some of these cases had been previously looked on as poliomyelitis. Two of the eight cases reacting negatively were unquestionably birth injuries. Of eight cases of chorea tested (following and including the first positive case whose history is given later), two gave positive Wassermann reactions. In three of the negative cases there was a definite history of rheumatism, while no history or evidence of rheumatism was noted in the two positive cases and in the three remaining negatively-reacting cases.

Mental deficiency was usually associated with some other condition as epilepsy or cerebrospinal syphilis. During the period covered by our cases there were thirty-three cases of mental deficiency admitted to the wards of the hospital of whom fourteen were syphilitic. This is a very much higher percentage than is usually found and does not express the true relation of mental deficiency due to syphilis to mental deficiency in general, but is the result of admitting cases for this particular study.

Among the more unusual lesions were an aotitis and a torticollis. Three patients with indefinite pain of obscure origin—once in the abdomen and twice in the legs—which had persisted irregularly for some time and for which no cause could be found, gave positive Wassermann reactions and the pain disappeared under specific treatment. The cases of acute arthritis were very interesting. Some patients were sent to us for tuberculosis and others for acute articular rheumatism. One case (knee) developed in a child with a tuberculous hip who had been under observation and treatment in the orthopedic department for several years.

The absence of Hutchinson's teeth which is so generally described as a common symptom of syphilis was very noticeable in our cases. In forty-eight patients who were old enough to have permanent incisors the teeth were notched in but three instances. Ill-shapen and deformed teeth were very common. They were usually small and peg-shaped with broken, irregular surfaces, and were separated widely from one another and without good alignment. We have also observed typical Hutchinson's teeth in a non-syphilitic patient.

It will be noted that most of our cases fall into one of three large groups. The first of these is made up of eye conditions (24 per

cent.) and is so well recognized that it needs no discussion. The second comprises lesions of the central nervous system (43 per cent.). So many of these cases have given positive Wassermann reactions when there has been nothing to suggest lues in the early or family history that we now make the test as a part of the routine examination of nervous cases. The third group is in many ways the most interesting as it contains a group of cases less frequently recognized as of a possible syphilitic origin. It is made up of cases of chorea, acute arthritis, torticollis, and obscure muscular pain—in other words clinical conditions which are often grouped together as "rheumatic"—and amounts to about 20 per cent. of our cases. It has long been recognized that these acute "rheumatic" conditions are the result of bacterial toxins, and our cases would indicate that syphilitic infection of an hereditary nature is one of the common causes of these cases developing in childhood. We have checked the diagnosis of these cases by repeated blood tests and by Wassermann reactions on the parents. The strongest proof that they are the result of syphilis and not an accidental association lies in the rapidity with which the lesions improve under antispecific treatment.

TREATMENT

The treatment we have gradually evolved consists of a combination of neosalvarsan and mercury. We prefer neosalvarsan to salvarsan because of the simplicity of the technic, which is an essential factor in dealing with children. The neosalvarsan is dissolved in 1 c.c. of freshly distilled water for each decigram and injected intravenously into any available vein with an ordinary glass hypodermic syringe. The entire preparation of the drug and syringe and the administration can be accomplished in from twelve to fifteen minutes. We made a number of intramuscular injections earlier but soon gave them up because of the pain and discomfort to the patient and the objections of the parents. The dose of the neosalvarsan varies according to the age and clinical condition of the patient—as a whole we give larger doses than are usually advised and have had no unpleasant or bad results. At first the injections were followed in a few instances by nausea and vomiting which we are inclined to attribute to the use of a "sterile" water instead of a freshly distilled water, as we have not observed such sequalae in the last year and a half. Although it is claimed by competent observers that neosalvarsan is less effective than salvarsan, we prefer neosalvarsan because it is effective and fulfills certain essential requirements for use with children which salvarsan does not. For the mercurial part of the treatment we have come to use gray powder exclusively in the late cases and to a very large extent

in the early cases. For the treatment of acute lesions (eruptions, ulcers, arthritides, etc.) the neosalvarsan is far more effective than inunctions of mercury, while the gray powder gives very good results and can be given over longer periods of time with less disturbance than mercury in any other form. We have had no experience with the mercurial injections which are so effective in the acquired forms of syphilis, as intramuscular injections are not practical with children, at least in the class of patients that we are treating.

The course of treatment we use varies somewhat, but with the usual case presenting an acute lesion is in general as follows: Three or four intravenous injections of neosalvarsan are given with a gradually increasing dosage two or three days apart. Then mercury is started, with small doses, which are gradually increased until the patient is taking a fairly large dose. In a few weeks this is interrupted for a short time and then repeated. In a number of cases a second and a third series of neosalvarsan injections are given alternating with the mercury. The mercurial treatment is continued for an indefinite period of time according to the requirements of the individual case and in general until a negative Wassermann reaction is obtained, if this is possible.

Forty of the seventy-four cases have been treated in this way and observed for a period of from three months to two years. As the cases differ so much in the types of lesions and length of treatment, they cannot be summarized to any advantage and the results of treatment are best seen by abstracting a few typical cases illustrating different points. We have given over two hundred intravenous injections of neosalvarsan and have never observed any bad or unpleasant effects besides those mentioned above.

EFFECT OF TREATMENT ON ACUTE LESIONS

There can be no question as to the value of neosalvarsan in acute syphilitic lesions with the exception of interstitial keratitis. In the acute joint lesions, ulcerations, general convulsions, torticollis, etc., the effects seem immediately to follow the first intravenous injection.

L. B., No. 3725, a girl of 17 months, was admitted for backward development. She had been normal until two months previously when she had an attack of convulsions. At the time of admission she was unable to stand alone and was apparently blind, not even noticing the milk bottle or a light. Attacks of general convulsions were frequent. Wassermann reactions on the blood and spinal fluid were positive. Neosalvarsan was given 1-14-13, 0.5; 1-17-13, 0.3; 1-22-13, 0.2 intramuscularly. The child was much better by this time and was beginning to notice objects. No attacks of convulsions since first injection. 1-31-13, 0.2, intravenously; 2-4-13, Wassermann positive; 2-12-13, 0.15, intravenously; 2-20-13, 0.2, intravenously.

Beginning about the first of February, 1913, there was rapid improvement and by the middle of the month the child was playing almost as a normal

infant. She was put on gray powder and on March 27 was discharged with a negative Wassermann. On April 24, one month later, there was a sudden return of symptoms and two days later she was readmitted. Between 12 o'clock noon and 9 p. m. there were ten attacks of general convulsions. She was then given 0.2 neosalvarsan intravenously which immediately stopped the convulsions. Inunctions of mercury were pushed for a month but no more neosalvarsan was used as the patient remained free from convulsive attacks. On May 17 the Wassermann reaction was negative and the patient was again discharged. In March, 1914 (fifteen months later), she was apparently normal although there have been "slight attacks of convulsions at times." She has taken gray powder intermittently.

R. C., No. 5043, a girl of 12 years, was admitted with a maniacal type of chorea about three weeks after the onset. She was unable to stand or walk and could not sit on a chair unless supported. Restraint was even necessary when she was in bed. Speech had been lost for several days. A family history of syphilis together with Hutchinson's teeth in the patient gave the idea of an underlying luetic infection. A dose of 0.4 gm. neosalvarsan was given a few hours after admission and a marked effect was noted within forty-eight hours. It was repeated two days later when a Wassermann reaction was strongly positive. Inside of a week the patient was up and about the ward and had lost most of the choreic symptoms. Neosalvarsan and mercury were continued for some time.

L. D., No. 4072, a 14-year-old girl, was admitted for acute pain and swelling of the right knee. One week later the ankle became swollen. A Wassermann taken two days previous to the involvement of the ankle was strongly positive. Neosalvarsan, 0.5, was given and in two days the swelling and pain had disappeared.

N. D., No. 3715, was admitted for an interstitial keratitis of five weeks' duration and an acute arthritis of the knee of two weeks. Wassermann, three plus positive. On Dec. 29, 1912, an injection of 0.9 neosalvarsan was followed by rapid improvement of the knee, but in a few days it became swollen and painful again. On Jan. 1, 1913, 0.9 neosalvarsan was given and followed by rapid relief. Treatment was continued with no return of the arthritis, but the interstitial keratitis showed but little improvement.

E. G., No. 6489, 8 years, admitted March 2, 1914, for acute arthritis of the right knee. Wassermann on March 5 was four plus positive. Neosalvarsan was given on March 7 and by March 11 all pain was gone from the knee and motion was good. She was given more neosalvarsan and discharged. Six weeks later the patient returned with an acutely swollen and painful left knee and the Wassermann was still four plus positive.

T. B., No. 3791, a boy of 6 years, was admitted with an extensive laryngeal ulcer of three weeks' duration. He was given five injections of neosalvarsan and within two weeks the ulcer had healed.

From these few reports the rapid action of neosalvarsan may be seen when the lesion is acute. The exception is apparently an acutely developing interstitial keratitis. In this condition it has an influence in some cases, but in our experience a long and extensive course of mercury is necessary in order to obtain much improvement.

TREATMENT AND CHRONIC LESIONS

We have been able to secure but little permanent effect from neosalvarsan nor have we observed any advantage from its use in chronic cases of the late type; that is, where a lesion such as a cerebrospinal syphilis or an interstitial keratitis has been present for

some time. As a matter of fact the most intensive treatment with combined neosalvarsan and mercury has been of little avail in these cases.

H. W., No. 3492, a boy of 14, was admitted for cerebrospinal syphilis (fixed unequal pupils, staggering gait, dementia, etc.), which was said to have first shown itself three years previously. Wasserman four plus positive. He was given 0.5 neosalvarsan in October, 1912, which was followed by inunctions of mercury. An injection of 0.9 neosalvarsan was given in December and the inunctions continued. In February, 1913, he was given two injections of neosalvarsan of 0.5 each and then gray powder was continued for several months. It was without effect on either the clinical condition or the Wassermann reaction. From time to time he has taken gray powder in large doses, but his condition to-day is worse than when treatment was started over twenty months ago.

In this same group must be placed the cases of hemiplegia following vascular changes and other conditions where structural changes have occurred. The only possible result of treatment in such cases is the prevention of further lesions.

TREATMENT AND THE WASSERMANN REACTION

It is not within the scope of this paper to enter into a theoretical discussion of the Wassermann reaction. For clinical purposes we look on the Wassermann reaction as a symptom of syphilis, comparable to an arthritis, a keratitis, a periostitis, etc. It is the most common symptom and present in such a large percentage of all clinically positive cases that we are inclined to be skeptical about the diagnosis of late syphilis in any untreated patient who has a negative Wassermann reaction. For example, we have had a case of interstitial keratitis in a boy who had marked Hutchinson's teeth. The Wassermann was repeatedly negative but because of the two classical symptoms of hereditary lues he was given a thorough antisyphilitic treatment which, however, was without effect. Later a rapid and marked improvement of the eye condition followed the use of tuberculin and in our opinion there is no reason for looking on this case as one of syphilis. A positive Wassermann reaction may be the only symptom of syphilis for many months or years (latent hereditary lues) and then other symptoms develop, as is shown both by the early history of some of our cases and the tests we have made on brothers and sisters of patients with manifest lues. It is quite difficult to get these patients with latent cases to take a course of treatment, but there is no reason why this symptom should be treated any less vigorously than a manifest lesion. Nothing definite is known of the future of these latent cases with a positive Wassermann and we are attempting to follow some which have not been treated.

The Wassermann reaction is stronger in hereditary lues than in any other form of syphilis and hence is correspondingly more difficult to make negative. Not only is this so but it is very apt to become positive again if treatment is stopped when it has been made negative as the result of treatment. We have not determined quantitative effects of the treatment on the Wassermann reaction.

N. D., No. 3715, a girl of 8 years, was admitted in December, 1912, for interstitial keratitis and acute arthritis. Wassermann four plus positive. The arthritis improved rapidly following two injections of neosalvarsan of 0.9 each followed by one of 0.5 and another 0.4. Gray powder, gr. 1 three times a day, was then given for three months when inunctions were started and continued for several weeks as the eyes did not improve. In May, 1913, the Wassermann was still four plus positive. Gray powder has been taken almost continuously since at the limit of tolerance and at times potassium iodid has been given. On Jan. 16, 1914, the Wassermann was still four plus positive and again on May 19, although the child has been given an intensive and uninterrupted antisyphilitic treatment for eighteen months and the eyes have improved considerably.

C. M., No. 53166, aged 13 years, was first seen on July 16, 1913, for an interstitial keratitis which began one month previously. Wassermann four plus positive. He was given 0.5 neosalvarsan intravenously every other day for three doses and then put on gray powder. Three months later the Wassermann was still positive and the gray powder was continued. On March 6, 1914, the Wassermann was still four plus positive, although the eyes had improved very much with eight months' continuous treatment.

K. V., No. 4081, a boy of 7 years, with an acute keratitis, had a positive Wassermann on Sept. 23, 1912, at which time he could not distinguish fingers at 5 feet. He was given neosalvarsan 0.3 and inside of a week was able to make out pictures across the room. Two more doses of neosalvarsan were given, but as on March 14, 1913, the Wassermann was still strongly positive. three more injections of 0.4 each were given. A Wassermann taken five weeks later, or May 2, 1913, was still four plus positive. Gray powder — 1 grain three times a day — was then given for three months but was without effect on the Wassermann. Eyes almost entirely well. This Wassermann, Aug. 1, 1913, was ten months after beginning treatment. The gray powder was then pushed to a point of salivation and kept up almost continuously until a *negative* Wassermann was obtained on Jan. 5, 1914, after fifteen months of vigorous treatment. Treatment was discontinued for two months and then a course of mercury given for one month. On May 19, 1914, six weeks after this was finished, the Wassermann reaction had again become *positive*.

At times it is not difficult to obtain a negative Wassermann reaction as in the following case.

G. G., No. 3443, an infant of 18 months, who was admitted for ulcers of the face and fingers (gummata) had a positive Wassermann Oct. 15, 1912. There was extremely rapid improvement following an injection of 0.22 neosalvarsan into the buttocks. This was repeated in fifteen days and the infant discharged one month later with a small nodule in the cheek the size of a pea. No other treatment was given. Five months later a Wassermann was taken which was strongly positive and the patient was given three intravenous injections of neosalvarsan of 0.25 each and discharged as there were no clinical lesions. Eight months later — fourteen months after first admission — the Wassermann was negative and in April of this year there had been no return of symptoms. This was one of the few cases with a permanent result following neosalvarsan alone and is decidedly exceptional in our experience.

In a treated case or a case under treatment in which the Wassermann has become negative, other symptoms of syphilis may develop, showing that the disease cannot be regarded as cured or quiescent from a negative Wassermann reaction alone.

E. P., No. 3995, a boy of 4½ years, was admitted for paronychia and malnutrition. No history obtainable, but the Wassermann was three plus positive on March 7, 1913. He was given seven intravenous injections of neosalvarsan (2 gm. in all) between this date and April 23, 1913. Inunctions of mercury were given to the point of salivation. There was marked improvement following the first few injections of neosalvarsan. In June and again in September he was given a course of mercury and on Oct. 24, 1913, (seven and one-half months after the first Wassermann) the Wassermann reaction was negative. In March of the present year he developed an alopecia which has become quite extensive. A Wassermann reaction on April 11, however, was negative, and again a few weeks later after a provocative dose of neosalvarsan.

SUMMARY

We have found the incidence of manifest hereditary lues of the "late" type much greater in proportion to the incidence of "early" syphilis than previous figures would indicate. The largest group of our cases (43 per cent.) exhibited lesions of the central nervous system, but we particularly wish to call attention to a group of cases with lesions associated as "rheumatic"—chorea, acute arthritis, torticollis, myalgia. By Wassermann reactions and the "therapeutic test" we have found that such lesions are not infrequently due to an hereditary syphilitic infection.

It has been our experience that acutely developing lesions respond promptly to intravenous injections of neosalvarsan, but that in order to obtain any permanent results and prevent the recurrence of symptoms an intensive and long continued mercurial treatment must be given in addition. The syphilitic infection—as measured by the Wassermann reaction—is most persistent, and although an intensive and uninterrupted antisyphilitic treatment has been given for nearly two years we have not been able to obtain a negative Wassermann reaction in some of our cases. Moreover, the Wassermann reaction has usually returned when treatment has been interrupted in those cases in which it has become negative as the result of treatment. We do not feel that it has been demonstrated as yet that an hereditary syphilitic infection of the "late" type can be eradicated.

St. Louis Children's Hospital.

ONE HUNDRED AND FORTY-ONE CASES OF RECURRENT VOMITING IN PRIVATE PRACTICE*

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The cases reported have been seen in private practice during the past eight years. Cases seen in consultation with other physicians have not been included.

The history diagnosis was based on definite periodic attacks of vomiting, for which condition the patient was brought to me.

Recurrent vomiting, in a vast majority of the cases, we found occurs in the offspring of those who for two or more generations have not been occupied with manual work. The recurrent vomiting patients are the offspring of those who have had business or professional occupation.

SUMMARY OF THE FEATURES OF CASES SEEN

Average time cases were followed was 104 weeks.

Sex: Seventy were boys; seventy-one were girls.

Family History: Rheumatism in one or both parents in forty; sick headache and bilious attacks in forty.

Appetite: In 31 per cent. was normal; in 69 per cent. it was indifferent; in 59.5 per cent. there was constipation.

Previous Feeding in 120 Cases: Twenty-one patients were nursed by mother nine months; forty were nursed by mother six months; four cases had been wet-nursed; sixty-nine had been difficult feeders on cow's milk and had been given the usual trial of foods.

Recurrent colds had been present in 41 per cent. This information was usually volunteered. In twelve there was definite history of eczema; six had habit tic. In fourteen there had been or was enuresis. In thirteen there had been rheumatism.

Urine: Nothing was known as to the condition of the urine in the attacks before coming under observation. The presence of acetone in the later attacks was noted with very few exceptions, but there were exceptions. A child in an attack may show acetone and in the next attack the urine may be free.

Onset of the Vomiting Seizures: Thirty-seven occurred during the first year; twenty-four occurred during the second year; twenty-one occurred during the third year. Onset in the youngest was 6 months. In three the onset was during the eighth year.

Duration of Interval Between Attacks in 119 Cases: Under 2 weeks, 22; from 2 to 4 weeks, 27; from 4 to 6 weeks, 10; from 6 to 12 weeks, 42; from 3 to 6 months, 40.

Duration and Severity of Seizures: The severity of the attacks varied widely; in some the child would vomit but two or three times. These usually represented the short-interval cases. In others the vomiting was protracted and severe. The average was three and one-half days. The longest seizure under observation continued thirteen days.

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*Read at the annual meeting of the American Pediatric Society, Stockbridge, Mass., May 28, 1914.

The average interval in 118 cases was eight and one-fourth weeks.

Temperature: 102 F. or over in 30; 103 F. or over in 10; 104 F. or over in 6; 105 F. or over in 3. In the remainder there was an elevation of from 100 to 102 F.

In taking the history in fifteen cases, the mother or attendants volunteered the information that the child had a peculiar breath at the time of the attack which we interpreted as the so-called "acetone breath."

Onset: Average age in 121 cases was 2 years and 8 months; average number of attacks in 99 cases had been 9; average age brought for treatment was 5 years.

Results: Of cases in which there were no attacks after beginning treatment: Forty-one were followed for from two to eight years. Of these forty-one, twenty-six were followed from three to eight years and fifteen were followed from two to three years. Fifteen were followed from one to two years; nine cases were followed from six months to one year; eighteen cases were followed less than six months. Several of the latter are recent cases in which decided gain in weight and general improvement has occurred. In nine cases there was very much improvement; the attacks were mild and comparatively infrequent. Of these eight were followed for from two and one-half to eight years; one was followed eighteen months. In thirty-three cases there was improvement; the attacks less frequent and less severe. Of these thirteen were followed from two and one-half to eight years; six were followed from one year to eighteen months. The remaining fourteen of this group were followed for varying periods under one year, but the records show that a decided impression had been made on the seizures.

In sixteen cases there was no apparent improvement. In some the treatment was indifferently carried out and the patient was dismissed. In others, no apparent impression was made on the disease and the seizures continued as before.

I have an unimproved case that has been under treatment for three years without benefit. The attacks continue to be very severe at from ten to twelve weeks' interval. I only recently was successful in having massage and exercise treatment instituted.

Another boy now aged 6 has attacks of either periodic fever, bronchitis or recurrent vomiting, rarely going for three months without an explosion of some kind. He has been my patient since he was 6 months of age.

In a vast majority of the cases the recurrence will be completely controlled if continued family cooperation is secured. A diet and living regimen will be followed faithfully by some parents as long as they can be kept anxious about the patient. When the child has passed three or four vomiting periods, the use of forbidden articles of food is often gradually resumed; the former methods of living are more attractive than the regimen laid down.

The association of recurrent vomiting with other forms of illness is interesting. As mentioned before, 41 per cent. of these children were subject to repeated bronchitis, usually of the spasmodic type, and not infrequently the bronchitis and the vomiting occurred simultaneously. Acetone was not present in these cases unless gastric symptoms were present. In others, the attacks will be distinct and separate. In one

patient there was in nearly every attack an acute spasmodic laryngitis; bronchial asthma very urgent followed and this again was followed sometimes, but not invariably by repeated vomiting. This child had suffered severely with eczema when an infant. Two boys, brothers, were of particular interest in that they had the attacks simultaneously, beginning with tonsillitis, followed by bronchitis and asthma and ending with recurrent vomiting lasting for a day or two. In a few cases the recurrent vomiting attacks were replaced by high fever for three or four days, with acetonuria and without other symptoms.

Two girls, aged 5 years, both in wretched condition, had mild vomiting every week or ten days. In both the acetone breath and acetone urine were continuously present.

The only fatal case seen by me and not included in the above statistics was in a girl 7 years of age living in a New York suburb. There had been previous attacks at about from three to six months' intervals. This attack was particularly severe, no fever but frequent and violent emesis. The vomiting had continued about four days when I saw her. At this time there was considerable exhaustion, but the child was fairly bright, answered questions and talked freely. I endorsed the attending physician's treatment, made a favorable prognosis, and the child died in five hours. Apparently, death was due to respiratory paralysis.

Three patients had been operated on for appendicitis in Paris without relief. A normal appendix was removed in each case. In three it was necessary to resort to morphin hypodermatically to control the vomiting seizures.

MANAGEMENT DURING THE INTERVAL

The management in the main was the same in all, and one reason for reporting only the cases seen during the past eight years is that during this period practically the same interval management has been carried out.

Diet: If the case is a pronounced one, the patient is given a diet with few restrictions, except that cow's milk, butter, cream and sugar are omitted. One egg is allowed perhaps every third day. Saccharin is permitted as a sweetening agent in some and very little sugar in any case.

Three meals daily are allowed with nothing between meals. Red meat is given scantily three times a week. Poultry and fish are given at other times. In some skimmed milk is allowed scantily, never more than one pint daily, often less. Puddings are made with skimmed milk. A grave error in our management of many children is the free use of cow's milk, butter, ice-cream and sugar. I could present dozens of records showing surprising gain in weight and marked improve-

ment in the general well-being of the patient after a considerable withdrawal of milk, cream and sugar from the diet.

The period of lactation in the human being is at the most a year and then the child is ready for other food than milk. I believe that the average well child would thrive far better if he were to get not more than one pint of milk daily after the fifteenth month. Sugar was not used except as a condiment as we now use honey, until three hundred years ago. Unknown millions lived their span without it.

Medication: The further treatment consists in the internal use of salicylate of soda, bicarbonate of soda, independent or in combination as advocated by Rachford. In a pronounced case I give 5 grains of sodium salicylate with 10 grains of sodium bicarbonate three times daily at five-day intervals, or 20 to 30 grains of the sodium bicarbonate daily for a month or two at first. This drug treatment is carried on with rest periods for months and years as the case may require.

Bodily Exercise: A very important factor in the management is in arranging daily physical exercise, such as riding horseback or the bicycle and walking so many street blocks a day. A warm bath and a brisk rub are given at night, and last but not the least important feature in the treatment is the use of physical therapeutics. In severe cases I use if possible daily massage together with various body manipulations and exercises, the latter sufficient to make the child perspire, but not to the point of exhaustion. The advantage of this phase of the treatment was called to my attention by Dr. W. P. Northrup.

Bowel Function: A daily evacuation of the bowels is insured by suitable measures.

Some children I take out of school. For others I advise a modified rest-cure, which means in bed until 10 a. m., rest one and one-half hours after dinner and in bed and lights out at 6 or 7 p. m. The nervous element in these cases is not to be forgotten. If the attendant is not agreeable to the patient her services are dispensed with. In some a temporary elimination of the mother has been of assistance. The precipitation of an attack by fatigue and fright is not unusual.

The regulation of the life and habits aids materially in the management, but is of little or no use if the carbon content in the food is not reduced to the oxidizing possibilities.

MANAGEMENT DURING THE ACUTE VOMITING ATTACK

I find that in the vast majority of the cases, a weak solution of sodium bicarbonate is best retained in the strength of 5 grains of the bicarbonate to 8 ounces hot water. This is given freely.

As laxatives the magnesia preparations are best retained and are used when a laxative is required. Calomel or mercury with chalk will

often increase the vomiting. In other cases their use supplies very material assistance. When the vomiting has continued for twenty-four to thirty-six hours the patient is given colonic flushings with sodium bicarbonate, 2 drams to 8 ounces of water at eight-hour intervals. The solution is best retained if it is given warm (105 F.), the tube inserted from 8 to 12 inches.

Feeding: Nothing is gained by attempts at forcing the feeding. When the child is ready for food, he is given barley or rice gruel with dried bread crusts or unsweetened zweiback.

Judging from the results obtained through the withdrawal of highly energized foods and in the use of active and passive exercises it would seem that the chief error in most cases rests in a defective oxidation, or in the giving of food substances of high carbon content in excess beyond the powers of normal oxidation.

In this connection the views of Francis Hare[1] are particularly interesting. Hare states that carbon intake must be offset by carbon expenditure or energy intake with compensating energy expenditure, if "hyperpyremia" (excess of fuel in the blood) is not to occur. When physiologic functions are deficient in maintaining a balance, and an excess of carbon or hydrocarbon is not wholly applied in the manufacture of additional fat, bile, milk or other secretion, or lost by exercise or menstruation, a pathologic function is then necessary to free the system of its excess of fuel. As such so-called pathologic functions, Hare mentions gout, migraine, gastralgia, bilious attacks, epilepsy and asthma, all of which operate through either a reduction of income or an increase of expenditure by vomiting, muscular action or fever, periodic in character. These attacks of migraine have been observed alternating in the same individual with attacks of gout; gout has subsided with the development of glycosuria, and women (according to Garrod) show greatest liability to the development of gout shortly after the menopause, one decarbonizing process replacing another.

I am not convinced of the reliability of this theory in its entirety, but find it peculiarly in harmony with the clinical facts under presentation, and (while not forgetting for a moment that proteid anaphylaxis (Meltzer) and reflex neuroses from various abnormalities are still to be considered as operative causes in such affections as asthma, recurrent vomiting and migraine) I wish to emphasize the following suggestive points:

Eczema, spasmodic laryngitis, cyclic vomiting and recurrent bronchitis and asthma are all notoriously frequent in children of gouty or rheumatic ancestry.

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1. Hare, Francis: Carbon Factor in Gout, Med. Rec., June 17, 1905.

All these conditions are met less frequently and with diminishing severity at the age of puberty when the processes of combustion and tissue building are at their maximum.

While two or more of these so-called pathologic functions are not ordinarily observed simultaneously in the same child, not a few children suffer from a number of these conditions in alternation over a period of years. In other words, the processes seem to be mutually compensatory.

In the winter months, when activity is lessened and perspiration is least, every one of the conditions mentioned is intensified.

Finally, in every instance, diet (as the cases show) is a most prominent factor influencing the susceptibility of the patient.

The statistics as presented were prepared from my case histories by my associate, Dr. Frank Elmer Johnson, to whom I wish to express my indebtedness.

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MYATONIA CONGENITA OF OPPENHEIM *

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Since Oppenheim[1] has described and outlined the clinical picture of this disease, a number of subsequent observations by this author and others have broadened the clinical aspect, adding new features to those first described.

Some observations were made, in which not only the muscles of the extremities were involved but also those of the neck and back; and in a series of cases the flaccid pseudoparalysis involved not only the musculature of neck in addition to the more commonly affected extremities, but also that of the whole trunk simultaneously, so that with the exception of the diaphragm all muscles supplied by the spinal nerves appeared paralytic.

The assertion that the muscles supplied by the cranial nerves remain intact in this disease had to be modified as the result of additional observations. A certain degree of participation of the cranial nerves especially of the facialis is in a few histories at least cursorily indicated. Pollak[2] published a report of a well-observed case with involvement of the musculature of the facial and hypoglossus nerves in addition to the affection of all the spinal nerve muscles with the exception of the diaphragm. Cases of facial affection are described by Tobler,[3] Collier and Wilson.[4]

REPORT OF CASE

The following case seen by me this year deserves here a more detailed description, not only because it adds to the small series last mentioned, but also for reasons later to be dwelt on:

History.—Second child, girl, born in America, April 5, 1914, of healthy Russian Jew parents. Their first baby died when 3 weeks old, presumably from cephalematoma, which possibly became infected, as baby was paralyzed before death. Family history negative; no intermarriage. Spontaneous normal birth at full term. Mother had felt quickening during this pregnancy as in the first. The new-born, according to a woman friend, cried loudly immediately after birth, and was able to move during the first week of life;

* From the Central Free Dispensary, Rush Medical College, Chicago.

1. Oppenheim: Monatschr. f. Psychiat. u. Neurol., September, 1900, viii; Berl. klin. Wchnschr., 1904, No. 10, p. 255; Lehrbuch der Nervenkrankheiten, Edition 5, Part 1, p. 242.

2. Pollak: Arch. f. Kinderh., 1910, liii, 373.

3. Tobler, L.: Ueber kongenitale Muskelatonie (Myatonia congenita Oppenheim), Jahrb. f. Kinderh., 1907, lvi, 33.

4. Collier and Wilson: Amyotonia Congenita, Brain, 1908, xxxi, 1.

the mother herself can make no statement on this point. She remembers at least, however, that in the second week of its life the baby moved only a little and very slowly and was completely paralyzed from the third week. The baby's cry was only a low, feeble one, the facial muscles showing no contractibility, "it cried only with its voice, but without its face." Abdominal respirations were labored. She was always a quiet baby. Stools were irregular, constipated, appetite and sleep good; breast-fed. These conditions did not change noticeably with the exception that the baby has exhibited during the last five or eight days a smile, and a slow, feeble mimical action of the face when crying.

Examination.—July 4, 1914: Girl, well nourished, face of healthy color. Panniculus adiposus well developed, skin warm, of healthy rosy tint. The baby lies on its back with all extremities immovable; when lifted up, they drop as lifeless to the pillow. The legs are rotated outwardly. Only very minimal slow, feeble movements of the fingers, toes and ankle-joints, spontaneously or on needle-pricks, to which the baby responds with very feeble crying. The abdomen is very prominent, tympanitic, the wall very flabby and soft. Respirations very frequent, from 70 to 80 per minute, irregular, abdominal, the diaphragm acting vigorously with marked inspiratory protrusions of the upper part of the abdomen and with retractions of the lower part of the thorax. The hand laid on the abdomen meets a marked resistance from the protrusion during inspiration. If the child is lifted up underneath the shoulders, the latter are pushed up to the ears on account of the flabbiness of the tissues; the arms and legs swing in complete flaccidity, the right arm thereby rotates into somewhat extreme position, as if without sufficiem hold in its shoulder-joint. In the other joints of the extremities there is to be noted no hypermobility. If the body is brought into sitting position, the head on account of weakness of the neck musculature wabbles and drops helplessly to any side into extreme positions as in a cadaver, the trunk falls forward, forming a marked kyphosis. The limbs are full and round. The tissues of the upper extremities are markedly flaccid, so that the muscles are not discernible from the panniculus adiposus; those of the lower extremities that are well developed, have an almost normal turgor; their panniculus adiposus is solid and the underlying musculature can be palpated only very indistinctly. There is nowhere any atrophy to be noted. Though the face shows good muscular contractions during crying, yet these occur with appreciable slowness, and in the quieter intervals the infrequency and feebleness of the mimic display gives to the face a certain immobility and blank expression. Smiling occurs only very rarely and very feebly. Both eyes follow a flame or bright object; in lateral positions the bulbs show horizontal nystagmus, when fixating. Pupils react promptly to light. The baby recognizes its mother. My finger introduced into the baby's mouth causes a vigorous contraction of the lips and pressure by the sucking tongue. Stools constipated, appetite normal.

Reflexes.—Patellar reflex, Achilles-tendon reflex, Babinski, reflexes of the abdominal wall and soles of the feet are entirely absent, biceps-tendon reflex doubtful. No fibrillary contractions. No mechanical irritability of the muscles of the extremities on tapping with the percussion hammer.

Electric irritability: The neuromuscular irritability, examined on the extremities and trunk with strong faradic current is entirely absent; also no response to single shocks. (Galvanic test see July 25.)

A gross lesion of the sensibility can be excluded; but one's impression is that crying is less on faradic irritation of the lower extremities than of the upper, and that the patient bears strong faradic stimuli easily.

Treatment.—Daily faradic treatment, massage and sea-salt bath.

Course of Disease.—July 21: Baby moves the left arm occasionally, being able slowly to bring the hand spontaneously even to the mouth, and hold a morsel of bread. Very rarely the right arm is moved, slowly and only slightly in the elbow. The toes and fingers seem to show a slight improvement of mobility, if there is any change at all.

July 25: Mother makes the statement that the baby can be induced to smile more easily. The crying is somewhat louder, and when quiet there is a beginning of a certain mimic display. Electric reaction: The examination of the muscles of the abdomen and the extremities reveals a complete lack of (neuromuscular) reaction on strong faradic current. The small muscles of the dorsum pedis contract moderately on strong galvanic current stimulation; quadriceps cruris and the muscles supplied by the peroneus nerve contract feebly. The contractions, however, are quick and short, not as in degeneration. The cathode closure contraction is somewhat stronger than the anode closure contraction.

July 29: During longer observation baby shows infrequency or almost entire absence of mimic display, but when pricked painfully shows good contractions of the facial muscles in crying. Horizontal nystagmus present especially in lateral positions of the eyeballs in fixating. The baby's legs are still motionless under ordinary conditions, but on several occasions they have been brought actively side to side in the bath, when previously they had been crossed over the shin-bone by the mother or passively bent in the knee-joint.

August 5: Baby is able to flex and somewhat extend the knee and hip-joints when in the bath, not otherwise. There is a decided improvement of both arms; occasional movements also are possible in both shoulder-joints, though less in the right one. Nystagmus. Crying with feeble voice. Respiration abdominal.

August 11: Condition the same: Crying very feeble. Spontaneous mimic display rare. Slight edema of dorsum pedis on both sides. The tissues of the legs are softer than before.

September 4: A moderate degree of flabbiness of the tissues of the legs has become evident; otherwise the condition is the same.

Summarizing the principal clinical features, we have flaccid pseudo-paralysis of the muscles of the extremities and the trunk with the exception of the diaphragm, and an involvement of the facial muscles, with a tendency toward improvement. There is absence of the faradic and diminution of the galvanic irritability of nerves and muscles, but no atrophy. Skin-reflexes and tendon-reflexes are absent.

By this symptom-complex this case can easily be identified with the clinical picture of Oppenheim's myatonia congenita whose features may be reproduced here, before I dwell on a few factors of my case that are worthy of special consideration.

SYMPTOMATOLOGY

The disease occurs in the earliest infancy without known causes. That the beginning may occur during the intra-uterine life is indicated by the frequent statement of the mothers that during pregnancy they had felt no movements of the child. The symptoms exist from birth and in most cases are noted during the first days of life. With attention lacking or inefficient, the conditions may escape the notice of the family for weeks or even months until the failure of the baby to learn to stand or walk reveals the motor disturbance, as is also often the case in congenital cerebral paralysis (paraplegia) or early poliomyelitis anterior. In a few cases, in which even under good observation the symptoms were not manifest at birth, various diseases as bronchitis,

pneumonia (Leclerc[5]) or diarrhea (Comby[6]) soon caused their appearance. In such cases we may consider these intercurrent affections partly responsible through their general weakening result, only in making the manifestations of myatonia more conspicuous. Yet Rosenberg[7] observed a case in which the weakness of the legs appeared as late as in the eleventh month; Collier and Wilson[4] report a similar observation and conclude that the disease may develop at any time in an apparently normal child in the first year of life.

The most conspicuous feature is the inactivity of the child and the flaccidity of the musculature. The affected extremities, the arms often and the legs invariably and in a greater degree, lie on the pillow as if paralyzed, because of the extreme weakness of the musculature; they remain motionless in the most uncomfortable positions in which they are brought during the examination. Perhaps the distal parts, namely, the fingers and toes, retain a slight spontaneous residual mobility or on needle-pricks. The movements in the other joints, should they be present, are slow and weak in the more pronounced cases, the muscular contractions being without energy and real locomotor effect. The limbs, lifted by the examiner, drop on the support as if lifeless. The affected muscles are as a rule markedly flaccid, hypotonic or atonic, of soft consistency, but not atrophic, as in poliomyelities anterior, or in neuritic paralysis; nor are fibrillary contractions to be observed.

It is impossible to distinguish by the touch between panniculus adiposus and muscles; the structures from the skin down to the bone seem to be one soft homogeneous substance. In a few cases, however, a certain degree of reduced volume of the muscles has been clinically noted, as by Batten, Collier and Wilson, Marburg and Schlivek; which fact does not surprise us, if we consider the inactivity of the muscles more or less absolute — apart from the nature of the disease. In other cases an abundant development of the panniculus adiposus renders it impossible to make a decision, and marked reduction of muscle with proliferation of connective and fat tissue has been revealed anatomically in cases in which the clinical appearances indicated well-developed muscles.

The joints may be loose, flaccid and more or less flail-like, so that the limbs are passively movable like loose appendages. Where the back and neck musculature is involved, the body, if brought to a sitting position, will fall forward and double up "as if made of rubber" with the formation of an extreme kyphotic curvature of the spinal column, and the head will wabble and fall to any side. Standing is impossible,

5. Leclerc: Gaz. d. hôp., 1907, No. 141.
6. Comby: Bullet. Soc. pédiatr. de Paris, October, 1907, p. 149.
7. Rosenberg: Ueber Myatonia congenita, Deutsch. Ztschr. f. Nervenh., 1906, xxxi, 130.

as the lower extremities collapse helplessly. Where the respiratory (intercostal) muscles participate, the respiration has lost its costo-abdominal character and becomes purely abdominal, the diaphragm acting vigorously in compensation. The weakness of the abdominal wall musculature manifests itself in more or less marked softness, protrusion and flabbiness of the abdomen and may cause constipation.

The reflexes are as a rule absent or diminished; likewise the neuromuscular irritability to both currents, but degeneration reaction will not be found. The sphincters of anus and bladder and the vasomotors are normal, the sensibility rarely (cases of Rothmann,[8] Collier and Wilson[4]) is slightly affected. The affection in its typical form is the antipode of Little's disease.

The course of the disease is chronic with a tendency toward improvement, a part of the more severe manifestations being capable of a gradual reparation during months or years, though perhaps in no case as yet has a complete recovery been observed. The improvement is mostly slow, to be of any amount, takes years, but even after a standstill for years, improvement can take place. The upper extremities apparently always recover first, and may gain even a normal function. The disease is not dangerous of itself, but it exposes the individual — as dystrophia musculorum progressiva — in a high degree to pulmonary affections, and their prognosis is markedly impaired through the weakness of the respiratory muscles. In fact, that pneumonia and bronchitis are among the chief causes of the high death-rate among these individuals is no mere accident. It is worthy of note that this clinical picture has not been observed in the adult, a fact partly explainable by the great dangers of accidental intercurrent diseases.

The more limited the atonia is, either from the beginning or as the result of improvement, the more limited and altered will be this picture of infirmity. Further attention should be given to the existence of rudimentary, abortive forms of the disease. From the literature we note the fact that considerable differences in the intensity of the symptoms undoubtedly exist. In some cases with hypotonia there were indeed only minor degrees of paresis. It is probable that some hypotonic conditions in infants may have a cause identical to that of the fully developed, pronounced pictures of myatonia congenita, especially if muscular weakness is manifested in the lack of energy of some movements.

NATURE AND PATHOLOGY

Oppenheim referred the myatonia to a retardation of development of the musculature, without excluding the possibility of a developmental retardation of certain nerve centers, especially of the ganglia

8. Rothmann: Monatschr. f. Psychiat. u. Neurol., 1909, xxv, Ergänzungsheft, p. 161.

of the anterior horns and their function. The possibility of a gradual development toward normal or almost normal function, but also of an aggravation — though very rare — is admitted.

The anatomic findings have been various; they place the affection near the group of the primary myopathias — dystrophia musculorum progressiva — which latter have a close relationship to the spinal myopathias; but clinical reasons so far do not allow us to classify myatonia with dystrophia progressiva. The most frequent alterations concern the musculature. The pathologic-anatomic findings of Spiller are in support of Oppenheim's view; the affected muscles were little developed amid an hyperplastic interfibrillary fat tissue; the muscle-fibers were very narrow, their longitudinal striation indistinct; the nervous system was intact.

Rothmann[8] observed a marked proliferation of the interstitial connective and fat tissues; the muscle-fibers were partly atrophic. Also Reyher and Helmholz found the muscles, especially of the legs, deeply affected; the fibers were partly thin, partly hypertrophic, their transversal striation partly indistinct, partly lacking.

In the cases of Baudouin[9] and Collier and Holmes,[10] the muscular changes resembled much those in primary myopathias. They observed also congenital hypoplasia of the cells of the anterior horns. In some of these cases the cells were markedly small and underdeveloped; or their number was decreased, but without any sign of inflammation; the peripheral nerves had defectively developed medullary sheaths, a condition which was considered a sign of underdevelopment.

In Rothmann's case there was extensive atrophia (*Schwund*) of the motor ganglia throughout the medulla spinalis. Also the white substance was not intact. The cross-section of the medulla spinalis was small *in toto*. The transverse section of the peripheral nerves was very narrow; there were also pathologic changes in the nuclei of a few cranial nerves (hypoglossus, vagus); these, however, had given no clinical symptom.

Thus really the affection may concern the whole peripheral neuron, the aplasia or hypoplasia involving in the one case this, in another case that segment of the neuron, including the muscle.

Bing,[11] differing from Spiller,[12] found a normal structure of an excised particle of muscle with the exception of perhaps a somewhat increased number of nuclei, and conceives the nature of the disease

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9. Baudouin: La myatonie congénitale (maladie d'Oppenheim), Semaine méd., 1907, xxxvii, 241.

10. Collier and Holmes: Brain, 1909, xxxii, 269.

11. Bing: Ueber atonische Zustände der kindlichen Muskultatur; vorläufige Mitteilung, Med. Klin., 1907, iii, 10.

12. Spiller: Myatonia congenita, Penn. Med. Bull., 1905, xvi, 342.

to be an inhibition of the development of the spinocerebellar nerve-tracts that regulate the tonus.

Vierordt thinks of a diminution of functional responsiveness for central and peripheral stimuli of the cells of the gray anterior horns of the medulla. The clonic spasms in the cases of Sorgente[13] would be explained by stimuli of maximum intensity.

Kaumheimer[14] draws the conclusion, from the histologic examination of a case, that myatonia is an entity which must be considered either as a toxic or endogenic affection, while the doctrine of inhibition of development or of inflammatory processes being the pathologic basis of myatonia must be discarded. The process has not necessarily found its completion at birth, but may cause alterations of the muscles even months later.

TREATMENT

The tendency of the disease toward partial or more pronounced spontaneous improvement renders it difficult to decide the contributing effect of any treatment; in addition to this, the descriptions of most cases in the literature represent merely snapshot pictures, so to speak. But it often seems as if a more pronounced improvement had taken place during treatment with electricity and massage, extended persistently over a long period.

COMMENT ON AUTHOR'S CASE

My own case is of great interest because of the involvement of the facial muscles, manifesting itself in lack or weakness of the mimic display; this was replaced by a certain degree of immobility, which improved before the other symptoms. The crying was markedly feeble, which fact finds its explanation in the involvement of the respiratory muscles, especially of the expiration musculature, a symptom also observed by Pollak,[2] Berti,[15] Baudouin,[9] Lugenbühl.[16] The high frequency of the respiration and the vigorous inspiratory protrusion of the abdomen point also to a weakness of the intercostal muscles, that show retractions during inspiration.

It is noteworthy that, though the pseudoparalysis was more marked and more persistent in the lower extremities, their turgor or tonus was almost normal for the first four months, while the entire upper extremities with their more visible improvement manifested a pro-

13. Sorgente: Due casi di atonia muscolare di Oppenheim, Pediatria, 1906, xiv, 358.

14. Kraumheimer: Jahrb. f. Kinderh., 1913, lxxviii, Ergänzungshaft, p. 170.

15. Berti: Contributione alla atonia muscolare congenita di Oppenheim. 3. adunanca della seziona Emiliana della Societa Italiana di pedriatria, Dec. 4, 1904; Pedriatria, 1905, p. 134.

16. Lugenbühl: Deutsch. med. Wchschr., 1907, p. 1439.

nounced atonia; the flaccidity of the shoulder-blade musculature permitted passive motions upward or medianward to the ears. While Cassirer[17] claims that in most cases the proximal muscles of the extremities are less affected than the distal ones, a number of cases have presented the reverse conditions (Kaumheimer,[14] Kundt,[18] Rosenberg[7]). To the latter group belongs also my case; only the fingers and toes were slightly movable at the first observation. The explanation of this may be that for the movements of small parts, such as the distal ends of the extremities, less strength is required. Similarly I could observe the first signs of improvement in the mobility of the legs while the baby was submerged in the bath, which diminished sufficiently the motion-inhibiting effect of friction and gravity for the at first only minimal functional capacity of the legs. The very obvious flabbiness of the abdominal wall and the softness of the tympanitic abdomen indicated an involvement of this musculature; there was also constipation. The neck and back musculature being involved, the pseudoparalysis concerned all the spinal nerve muscles with the exception of the diaphragm.

Nystagmus, as in my case, was noted also by Koch[19] and was referred by him to a congenital defect of eyesight. Whether this ocular symptom in my case should be considered as a manifestation of a congenital weakness of the external muscles of the bulbus, similar to that observed in lesser degrees of paresis of an extra-ocular muscle, or of a functional exhaustion, similar to the nystagmus in miners, or whether it should be regarded of central origin, cannot be decided with certainty. I am inclined to consider in my case the presence of weakness of extra-ocular muscles as a condition analogous with those of other muscles, affected in myatonia. A defect of eyesight in my case may be excluded by the fact that the child fixates well and recognizes persons, responding after improvement to the smile of the mother.

An interesting feature resulted from the electric examination of the nerves and muscles; though the irritability even to strong faradic currents was completely lost, there was nevertheless present a somewhat lessened irritability to the galvanic current; differing from degeneration reaction, however, the contractions were quick, short, though weak, and the cathode closure contractions were somewhat stronger than the anode closure contractions. As usually reported, the irritability of the muscles and nerves to the galvanic as well as the faradic currents in myatonia congenita of Oppenheim is found either absent or much decreased; in the case of Bing[11] the reaction was almost normal; in

17. Cassirer: Lewandowsky's Handbuch der Neurologie, 1911, ii, 230.
18. Kundt: Ueber Myatonia congenita, Inaug. Diss., Leipsic, 1905.
19. Koch: Jahrb. f. Kinderh., 1913, lxxviii, Ergänzungsheft, p. 305.

that of Muggia[20] there was normal neuromuscular irritability. No qualitative alterations of the contraction formula is to be observed; Cassirer, Oppenheim, Rosenberg and others never found degeneration reaction; Rothmann, however, saw a rather sluggish contraction in his case.

Collier and Wilson described a special form of alteration of the electric reaction, which they declared was characteristic of myatonia, and they termed it "amyotonic reaction," and Chène concurs in this view. According to the observations of these authors the muscles react to the faradic current with diminished irritability, while to the galvanic current not only are the formula and character of the contractions normal, but also the quantitative alteration is only moderate. This special reaction is declared to be demonstrable to a certain degree in every muscle, most markedly, however, in the muscles most affected by the pseudoparalysis. The reverse of this modus of electric reaction was encountered by Koch, viz., the decrease of irritability was more marked for the galvanic than for the faradic current.

There exists no differential diagnostic difficulty in our case. Poliomyelitis must be excluded by the congenital character, the absence of febrile beginning, of vasomotor anomalies, the absence of degenerative atrophy and degeneration reaction, and by the symmetry and extent of the motor disturbances. The muscles in poliomyelitis recover rather by groups and the stage of reparation is much shorter. Pseudoparalysis rachitica is out of the question.

The extreme rarity of acute generalized polyneuritis at this age militates against this disease, also the absence of an etiologic factor (diphtheria), of pain or sensible disturbances of a higher degree; the absence of disturbances of the heart-action and deglutition, and the lack of degenerative atrophy.

In dystrophia musculorum progressiva we would expect a later onset, possibly a hereditary factor, gradual progress, the presence of localized myatrophia, perhaps hypertrophic and pseudohypertrophic muscles and no participation of the distal muscles, especially of hand and foot, which is rare in dystrophia. In Werdnig-Hoffmann's spinal atrophy of muscles we would have steady progress, an exact localization of atrophic paresis (proximal distribution) and degenerative reaction.

4557 Broadway.

20. Muggia: Adunanza della sezione Piemontese della Societa Pedriatrica Italiana del febbraio, 1903; Pedriatria, 1903, p. 179.

REFRACTORY OR SO-CALLED "FAST" CASES OF MENINGOCOCCUS MENINGITIS*

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It is well known that since the introduction of the antimeningitis serum by Flexner in 1906, the mortality of meningococcus meningitis has decreased from 75 or 80 per cent. to 25 per cent. or less. Despite this fact, it is obvious that in a small percentage of cases the treatment of this disease has not yet reached perfection. That this small number of cases does not respond to the serum, in spite of its accurate and prolonged use, gives us food for thought and compels us to inquire into the causes of the occasional failure with this method of treatment.

CAUSES OF OCCASIONAL FAILURE

These causes may be grouped as follows:

1. *Fulminating Cases.*—The endotoxins of the meningococcus may be so virulent as to make neutralization by the antimeningitis serum impossible. The action of the serum is dependent on the presence of a number of immune substances, bacteriolysins or antibacterial substances, bacteriotropins (opsonins) and antiendotoxins. When those substances are not present in sufficient quantity or are not of sufficient potency to overcome the endotoxins of the invading organism, we have the fulminating cases which practically always end in death, despite early diagnosis and prompt treatment. It is questionable whether a serum of sufficient concentration or strength can be obtained to combat successfully this rapidly fatal type.

2. *Incomplete, Insufficient or Unskilled Use of the Antimeningitis Serum.*—It is needless to dwell on this cause of the failure of the serum to cure, for it is a matter of common knowledge that, in general, the mortality statistics are the lowest when based on the experiences of the most competent observers and workers in this field.

3. *Unstable Nervous System.*—In infants, lack of success is often due to the existence of an unstable and delicate nervous system and to the early development of acute hydrocephalus.

4. *Intercurrent Diseases.*—Complications having no direct connection with the disease, as tuberculosis or pneumonia, may produce a fatal result, in spite of the beneficial action of the serum.

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5. *Refractory Cases.*—Though few, these cases do occur and must be considered as constituting a distinct type. Although the meningococcus is found in the spinal fluid of these patients, the cases are little or not at all influenced by the injection of the serum. On that account they run a prolonged course of weeks or months, usually with high, septic temperature and occasional remissions in symptoms, until they finally result either in death or in a gradual protracted recovery. They are the "fast" cases, so called by Flexner, because they are supposed to be due to the existence of strains of meningococci which are fast to the serum employed.

It is only in recent years that fundamental biologic distinctions have been made between the various strains of the meningococcus. One strain is more resistant to solution by immune serum; another is more easily digested by the intraleukocytic enzyme; another more virulent when injected into animals — differences formerly never recognized. When this is taken into consideration, it is readily seen why certain cases do not respond favorably to the ordinary antimeningitis serum — they are produced by a strain of the organism which is refractory or fast to the serum used. They must not be confused with the type of cases described by Dopter, in which the clinical course is the same, but in which the infecting organism is not the meningococcus, but the closely allied parameningococcus, which possesses specific agglutinins and precipitins, does not react to the ordinary antimeningitis serum, but does respond beneficially to the serum prepared from its own cultures.

The following are reports of two refractory cases of meningococcus meningitis:

Case 1.—M. S., aged 6. There had been no previous illness. Two days before admission there had been headache, vomiting and apathy, these symptoms having increased in severity to the date of patient's entrance to the hospital. The physical examination on admission revealed typical signs of meningitis, apathy alternating with irritability, rigidity of the neck, scattered petechiae, Macewen, Kernig and Brudzinski signs, a tache cérébrale and a moderate temperature. Lumbar puncture revealed cloudy fluid which was under considerably increased pressure and which showed the meningococcus in smears and cultures. Flexner's serum promptly introduced into the spinal canal failed to result beneficially, and instead of a rapid recovery, as was expected, the symptoms continued unabated for about a month in spite of repeated injections, given almost daily for the first week, then somewhat less frequently. The temperature remained septic in type; the restlessness, headache and signs of spinal irritability continued as on admission, and the spinal fluid at each puncture showed the meningococcus. At the end of four weeks the child's condition began to improve and kept steadily becoming better until the patient was finally discharged well, fifty-eight days after admission. The last culture of the spinal fluid, taken twenty-six days before discharge, still contained meningococci.

Case 2.—J. H., aged 7½. The onset of this case was sudden, four days before admission, the child having complained of very severe headache and

pains in the neck and back. The patient had vomited several times and had had high fever. Physical examination showed the usual manifestations of meningitis — irritability, tache, Kernig, Babinski and Brudzinski signs and a petechial rash. On lumbar puncture 35 c.c. of cloudy fluid was withdrawn and found to contain the meningococcus. The fluid withdrawn at the third puncture, that is, after two injections of the Flexner serum, showed no meningococci in smears and in culture mediums. The symptoms, however, continued for five weeks. Repeated lumbar punctures were made, and although the fluid was always reported sterile after the second injection, the antimeningitis serum was injected on several occasions. The patient recovered after a protracted illness of nearly sixteen weeks.

It is interesting to note that these two refractory cases of meningococcus meningitis came from the same neighborhood, were infected at about the same period of time and failed to react to repeated injections of the antimeningitis serum.

To combat effectively these refractory cases illustrated by the above abstracts, it might be wise to culture the various strains of meningococci and to establish a "fast" antimeningitis serum to be used only in this type of infection, just as Dopter has prepared and used his antiparameningococcus serum; so that by this means we may eventually hope for a still greater diminution in the mortality of meningococcus meningitis.

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CLINICAL DEPARTMENT

PERSISTENT VOMITING ASSOCIATED WITH "RUMINATION" IN AN INFANT OF FIVE MONTHS

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A "vomiting baby" presents as many baffling problems as any condition in infancy. It is too often assumed that vomiting is a natural process and that it is only necessary to bide one's time and the baby will outgrow it. As a result of this attitude of mind, efforts at determining a cause of the vomiting are relaxed, matters are allowed to drift along, and medicines are given to satisfy one's conscience and to appease the family.

The most common causes of vomiting in infancy are connected with improper food and incorrect methods of feeding. A careful adaptation of the food to the child's tolerance or a proper regulation of the intervals of feeding and the amount of food in twenty-four hours will as a rule meet the indications in such cases. The question may reasonably be asked: How may "such cases" be determined? The obvious answer is: By a careful study and observation of the case under consideration. Too little attention is paid to the investigation of the conditions with which vomiting in infancy is related. Pitiful examples of this fact are seen in the cases of pyloric stenosis which are brought to the hospitals.

The following history represents an instance of vomiting connected with an unusual condition. The case is of sufficient interest on account of its rarity and of the results of treatment to warrant its being reported:

The baby was first seen on Oct. 1, 1913, when it was 5½ months old. It was a first child, full term, and a difficult forceps delivery. The weight at birth was 8¾ pounds. At 2 weeks of age the weight was 8¼ pounds, and at 5 months 8 pounds. She was breast-fed for four weeks. The mother was then taken ill and breast-feedings were stopped. On the breast the child gave no symptoms of vomiting until the end of the third week. During the fifth week she vomited after every bottle. The vomiting was not projectile in character.

The first artificial food, which was begun at four weeks of age, was: milk 6 ounces taken from the top 12 ounces of a quart; water 22 ounces; milk-sugar 7 teaspoonfuls; milk of magnesia 1 teaspoonful—2½ to 3 ounces every two hours. On this mixture, vomiting took place after every bottle. The bowels were constipated. This food was continued for three months.

The second food was condensed milk 1 part, water 8 parts. This was tried for three weeks. The vomiting improved somewhat. This food was stopped on account of an acute attack of indigestion.

The third food was peptonized milk. This was begun at four months of age. It was prepared as follows: milk 16 ounces, water 16 ounces, and a peptonizing powder was added. Vomiting continued on this mixture after every feeding.

The fourth food was malted milk, which was being given when the child was first seen. This was begun one week after the child was 5 months old. It was prepared as follows: malted milk three teaspoonfuls to 5½ ounces of water. The baby was fed every three hours and given 4 ounces of this mixture at each feeding. The vomiting was less for the first four days, but for three days past had occurred after every bottle. The bowels were constipated on this. The baby was hungry after each of its feedings.

The physical examination showed nothing abnormal, except that the baby was much emaciated. The abdomen was watched for peristaltic waves, but no waves were seen. The child was vigorous in spite of its poor nutrition.

No adequate explanation for the persistent vomiting was determined at the first visit. The case was regarded as one of gastric indigestion and a food low in fat was prescribed, and the intervals of feeding were increased from two and one-half to three hours. The mother was instructed not to handle the baby after feeding and to feed her at regular times. The child was put on a skimmed-milk mixture, made up as follows: skimmed milk 18 ounces, obtained by removing 4 ounces of cream from the top of a quart; barley-water 14 ounces; lime-water 2 ounces; cane-sugar 2 tablespoonfuls. Of this mixture, 5 ounces every three hours and six feedings a day were advised. The barley-water was made by adding a tablespoonful of barley flour to one pint of water and cooking the mixture for twenty minutes.

October 16 weight was 8 pounds. The vomiting showed no improvement on the skimmed-milk mixture. The baby was watched while taking its bottle, and the following phenomenon was noted. After finishing the feeding the baby began a suction motion with its tongue. She pushed out the tongue between the lips and drew it back again. This was repeated for ten or fifteen minutes. With each projection of the tongue between the lips milk came into the baby's mouth. She would swallow the mouthful and pump more out of the stomach. From time to time a large quantity would be drawn into the mouth and flow between the lips. This constituted the vomiting. The mother had noted this tongue-action from the time the baby was 4 months old and volunteered the information that she had often compared it to a cow chewing her cud. From the mother's statement and from the observation of the child, the descriptive word "rumination" fitted the phenomenon. At this date the baby was put on a mixture made up as follows: barley flour four tablespoonfuls, and milk one quart. This was to be cooked two hours in a double boiler. Of this mixture four tablespoonfuls were to be given every four hours, five feedings a day. After cooking, this mixture made up about one quart of food. After it was heated to feed to the baby, it was of the consistency of condensed milk. It was fed with a spoon. The baby was offered water between feedings and would take an ounce or two at a time. It is an interesting fact that the baby retained this without any evidence of the tongue-suction which followed the milk feedings.

October 23, the mother reported that the vomiting was less than on the skimmed milk. The mixture was changed by increasing the barley flour to six tablespoonfuls, which was added to one quart of milk. The object of the mixture was to obtain a food of such consistency that the baby could not pump it out of the stomach. Four tablespoonfuls of this were to be given every four hours, five feedings a day.

November 10, weight was 12 pounds. The baby had gained four pounds in three weeks. The vomiting and rumination stopped entirely as soon as the food was made up with six tablespoonfuls of barley flour to a quart of milk. The stools were three to four a day and were yellow, smooth and semi-formed. The baby was happy all the time and slept from 6:30 p. m. to

5:30 the next morning. She was getting four feedings a day of six tablespoonfuls at a feeding. The hours of feeding were 6 and 10 a. m., and 2 and 6 p. m.

November 24, weight was 12¾ pounds. One week ago she refused the food entirely for three days. She was not urged to eat and her appetite returned promptly. The vomiting and tongue-suction had not returned. There was now noted craniotabes of the right and left parietal bones.

One-half the yolk of a hard-boiled egg, mixed with the amount of barley-milk given at the 2 p. m. feeding, was added to the diet. The egg was to be boiled twenty-five minutes. One ounce of orange-juice was to be given at 8 a. m. The formula for making the gruel was now as follows: milk, 1½ quarts; barley flour, nine tablespoonfuls. The whole was cooked for two hours in a double boiler. Four tablespoonfuls of this mixture was given every four hours, at 6 and 10 a. m., and 2 and 6 p. m.

Feb. 18, 1914, at the age of 10 months the baby weighed 19 pounds and 14 ounces, net; head, 17½ inches; anterior fontanelle, 1¾ inches. Craniotabes of both parietal bones was still present. There were no other signs of rickets, except a rather prominent abdomen. The vomiting and tongue-suction had not returned. The child was very vigorous, slept well and was happy.

June 10, the mother reported that the child, aged 13 months, weighed 23¾ pounds, net. This was a gain of 15¾ pounds in seven months. There were five teeth. The child was sitting alone and trying to creep. She said a few single words, such as "dada," "mama," "kittie," etc. Her appetite was satisfied. She was having two stools a day. There had been no vomiting and tongue-sucking since October 16. The baby slept from 6 p. m. to 6 a. m. She was a happy, merry child, and seldom cried. She would often sleep from 10 until 2 o'clock in the daytime.

The following régime had been in effect for three months:

6 a. m.: Milk gruel, three tablespoonfuls.

8 a. m.: Orange-juice 2 ounces.

10 a. m.: Milk gruel, three tablespoonfuls; yolk of hard-boiled egg or cooked cereal (three tablespoonfuls). The cereal was taken without the addition of milk.

2 p. m.: Milk gruel, three tablespoonfuls; spinach, two tablespoonfuls; beef juice, 1 ounce with bread-crumbs.

6 p. m.: Milk gruel, three tablespoonfuls; yolk of hard-boiled egg.

The milk gruel was made as follows: whole milk, one quart; barley flour, five tablespoonfuls. This mixture was cooked in a double boiler for two hours. The spinach was cooked two hours and then rubbed through a fine sieve. The baby drank at least a glass (8 ounces) of water in twenty-four hours.

DISCUSSION

1. The vomiting in this case was apparently due to a peculiar suction-action of the tongue. The phenomenon may best be compared to that of a cow chewing its cud. The descriptive word "rumination" applies more closely than any other to this phenomenon. The consistency of the milk gruel was compared to that of glue. The question arises as to whether food of this peculiar character presented mechanical difficulties to its being pumped out of the stomach.

2. This is one illustrative instance of the tolerance of a 5-months-old baby for starch. No deduction is to be made from this single experience that all babies of this age can tolerate as large a quantity

of starch as did this baby. At the same time, there is good reason to believe that young babies can tolerate larger quantities of starch than have heretofore been given.

3. This baby tolerated a mixed diet of egg, cereal, fruit and green vegetables after 8 months of age. There was evidence of beginning rickets, which showed a decided retrogression after the mixed diet was begun. This suggests the advisability of introducing solid food into the dietary of infants earlier than is usually the practice. This suggestion applies particularly to healthy children.

4. A wider application of this form of dietetic treatment is suggested in cases of vomiting. It is necessary to exclude organic causes for persistent vomiting, for example such as pyloric stenosis. It is also advised to try first such dietetic procedures as food with low fat, lengthening the intervals of feeding and avoidance of handling after feeding, but there are many cases of vomiting which persist in spite of rational treatment. It is suggested that a trial be given of a food of the consistency which was given in the foregoing case. At what age may this food be tried? The patient in the case just cited was 5 months old. I have had no experience with it in babies younger than 5 months, but believe it may be applicable in younger babies.

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